

ACTA RADIOLOGICA

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OFFICIAL ORGAN OF THE RADIOLOGICAL SOCIETIES OF DENMARK, FINLAND, NORWAY AND SWEDEN

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DIAGNOSIS

SEVENTH SYMPOSIUM

NEURORADIOLOGICUM

NEW YORK

21-25 September 1964

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R.Y. Examinations - Angiography

ACTA RADIOLOGICA

OFFICIAL ORGAN OF THE RADIOLOGICAL SOCIETIES OF
DENMARK, FINLAND, NORWAY AND SWEDEN

Vol 5

DIAGNOSIS

1964

SEVENTH SYMPOSIUM NEURORADIOLOGICUM NEW YORK

21—25 September 1964

In this special issue we have the privilege of publishing the majority of the papers presented at the Seventh Symposium Neuroradiologicum in New York. A few of the papers read at the Symposium were not submitted for publication; the titles of these are included in Table of Contents. Communications dealing mainly with biologic and therapeutic aspects are published in Volume 5 of the series Therapy, Physics, Biology.

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Papers should not exceed 24 pages, including space for figures and tables. Only in exceptional cases will contributions requiring more space be accepted for publication in the journal. More extensive articles may be published as Supplements for which special conditions apply.

All contributions should ordinarily be addressed, to the *Editorial Secretary Acta Radiologica Box 2052 Stockholm 2 Sweden*. Papers from Denmark, Finland and Norway may for convenience be submitted to the Editors of the respective countries for preliminary revision. The name and address of the department or hospital at which the work was carried out should be given at the top of the paper; the author should add an address to which correspondence can be directed and retain a copy of the typescript for reference.

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BOJSEN E. and DAHN I. Selective angiography of bronchial and intercostal arteries. *Acta radiol. Diagn.* 3 (1965), 515.
KERTH A. Human embryology and morphology 1st edition, p. 523. Arnold & Co. London 1948.

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It has been the rule in these special issues of *Acta Radiologica* that all contributions read at the Symposium and presented to us for publication are accepted. As far as possible the same principle has been applied for this Symposium although in some cases the articles in the Editor's opinion deal with aspects of radiology that are already well known. The same principles with respect to editorial revision have been applied for these papers as for those in the therapy section. In some instances the wording of the titles has been slightly altered. The papers are arranged in alphabetical order within adequate subject divisions. Owing to the large number of papers and the revision which in some cases was found necessary the work of publication has required a longer time than was originally expected. Financial contributions towards the publication of this special issue have been received from the Treasury and Organisers of the Seventh Symposium and *Acta Radiologica* is deeply grateful for this assistance.

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PRESIDENTIAL ADDRESS

First let me thank you, Dr Ruggiero, for the great honor you have bestowed upon me in investing me with this chain of office. I shall do my best uphold to the traditions of these Symposia.

Ladies and Gentlemen

I take great pleasure in welcoming all of you to the VII Symposium Neuro radiologicum. It is my hope that you will enjoy not only the scientific aspects of the Symposium but also generally your visit to this great city of New York.

I have endeavored to offer those attending this meeting as interesting and varied a scientific program as was possible. I am grateful to the Program Committee for their faithful and valuable assistance in the selection of the papers on the basis of their scientific merit. In spite of their careful selection the number of scientific contributions (151) is still large. The number of papers presented at this Symposium however is only slightly larger than that of the VI Symposium in Rome at which 147 papers were accepted for presentation. At the II Symposium in 1949 there were 24 papers, at the III in Stockholm there were 32 papers and at the IV Symposium in London in 1955 there were already 67 papers.

In the earlier Symposia (particularly those of 1949 and 1952) the idea was expressed that these meetings should retain their original quality that is that they should be symposia and not congresses that there should be no set time for presentation of a paper or for the discussion that followed. Also the tradition is that a president is selected who is in complete charge of organizing the meeting without being hampered by rules and regulations. Unfortunately the first idea is no longer tenable. Knowledge is accumulating at too rapid a pace and interest in this specialty has grown to a point where it is not practical sufficiently to limit the number of contributors. Henceforth probably new criteria must apply. Papers can only be accepted if they truly represent new knowledge and new research.

Growth is inevitable and the increasing number of contributions is a manifestation of increasing interest. Evolution of thought is not only inevitable but also necessary if man is to continue to develop and expand his knowledge. And so it is that at the first three Symposia there were papers dealing only with radiography of the skull and spine, encephalography, ventriculography, cerebral angiography and myelography. There was one paper, however, at

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SKULL ABNORMALITIES ASSOCIATED WITH THE ARNOLD CHIARI MALFORMATION

by

E. KRUYFF and R. JEFFS

Children with a spina bifida often present signs and symptoms of bladder dysfunction — neurogenic bladder. It is also known that meningo myeloceles are frequently associated with the so called Arnold Chiari deformity of the hindbrain. It seemed of interest to investigate whether a radiologically demonstrable correlation between the spina bifida and possible skull abnormalities existed in these children.

Material In a period of 11 months 93 patients with a neurogenic bladder were sent for routine urography, to which was added routine skull radiography. Thirteen of them with a history of tumor of or trauma to the spinal cord were excluded from the series (their skull films were normal). Eighty patients had a meningomyelocele; their ages ranged from 1 to 16 years (Fig. 1) and these are our concern in this study.

Spinal defects: widening of the spinal canal and neural arch defects were constantly present but to a variable degree and location (Fig. 2), 20% had spinal dysraphism only in the sacral or only in the lumbar area while the majority had involvement of more than one segment of the spine. Diastema

the III Symposium devoted to the use of diiodofluorescein in the diagnosis of brain tumors. At the London Symposium a discussion on isotope encephalography was included, and a paper on the use of ultrasound to determine displacement of the midline structures was presented. The latter two methods of diagnosis received little attention and, I must admit, the preliminary results of isotope encephalography as reported at that meeting were not too encouraging. Since that time, considerable progress has been made in the field of isotope encephalography and, more recently, the biological uses of ultrasound are receiving increasing attention. While in the beginning ultrasound was used only to determine displacement of the midline structures in the brain at the present time we are attempting to obtain cross sections of the brain in all planes which will demonstrate the anatomy in each plane. This area offers exciting opportunities for future research. Radiotherapy of tumours of the central nervous system and radiobiology are receiving more attention in this Symposium.

It is my greatest desire and hope that the scientific program of this Symposium will convey to you an accurate idea of what neuroradiology is today.

I am happy to see that there are, distributed widely throughout the program, a number of papers dealing with the research aspects of neuroradiology. I do not see how neuroradiology can endure as a subspecialty of radiology unless the young men of today are stimulated to seek the truths that lie ahead. It is necessary, therefore, to develop and support independent research in neuroradiology.

Let me take this opportunity to express one last thought. I believe that specialization within radiology has become a necessity. It is generally agreed that it is necessary for the physician or surgeon to become acquainted with all aspects of diagnosis and treatment and, in fact, this should include all diseases. Specialization, however developed out of the need to provide more knowledge in depth. It is also important for the specialist to know all he can about every aspect surrounding his field. In the case of neurological surgery in the past the neurosurgeon served not only as clinical neurologist in most instances but also as neuropathologist, neurophysiologist, neuroanatomist and neuroradiologist as well as surgeon. Nowadays the fields have advanced to a degree where such generalization is no longer possible and — granting that we must know all we can about all aspects of our specialties — in order to continue to advance our knowledge it is necessary to concentrate our efforts in a given area to achieve the only thing that is worthwhile: excellence.

And with these thoughts I declare open the VII Symposium Neuroradiologicum.

Juan M. Taveras

SKULL ABNORMALITIES ASSOCIATED WITH THE ARNOLD CHIARI MALFORMATION

by

E. KRUMFF and R. JEFFS

Children with a spina bifida often present signs and symptoms of bladder dysfunction — neurogenic bladder. It is also known that meningo myeloceles are frequently associated with the so called Arnold Chiari deformity of the hindbrain. It seemed of interest to investigate whether a radiologically demonstrable correlation between the spina bifida and possible skull abnormalities existed in these children.

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Spinal defects widening of the spinal canal and neural arch defects were constantly present but to a variable degree and location (Fig. 2). 25 % had spina bifida only in the sacral or only in the lumbar area while the majority had involvement of more than one segment of the spine. Diastema

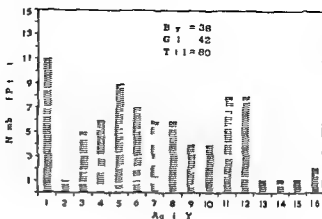


Fig. 1 Distribution of 80 neurogenic bladder patients

tomycia was seen 5 times. Rib anomalies were seen in 11 %, while hip dislocation was frequently encountered (40 %), in half of this number bilaterally. No attempt was made to classify other congenital defects.

The abnormalities were confined to the skull and particularly to the chondrocranium. These were 1) foramen magnum enlargement, 2) scalloping of the postero-medial aspects of the petrous bones, 3) flattening of the floor of the posterior fossa.

In 71 % of the patients a definite and symmetrical enlargement of the foramen magnum was demonstrated. The foramen was seen to be enlarged in all dimensions and often seemed rounder than normal, while in the region of the opisthion a small incisure was sometimes seen. The shapes of the foramen magnum encountered most frequently are shown in Fig. 3, in comparison with the normal foramen magnum (Fig. 4).

The bilateral dimension of the foramen magnum was taken at the largest width of the foramen as seen in the 10° half lateral view in the supine position. The length of the foramen was taken by measuring the distance between opisthion and basion in a special lateral projection of the foramen. The results of these measurements and those of normal patients taken for comparison are shown in Table 1. The ages as given are not inclusive.

The second abnormality, a rather peculiar and new one, was seen in the basal view. It consists of a scalloping of the postero-medial aspects of the petrous bones (Fig. 5). This was present in 65 %. At post mortem studies of the base of the skull, acquired from a group of infants with meningomyeloceles, which also showed this scalloping of the base in the films, it was revealed that this scalloping was produced by the configuration of a deep sulcus. This groove was running laterally from the non-displaced jugular tubercles and medially, posteriorly but also partially inferiorly to the petrous bones and at their

Table 1

Measurements of width and length in millimeters of the foramen magnum in normal and Arnold Chiari skulls (with complete and incomplete triads)

Normal						Arnold Chiari group					
Age in years	Sex	No	Width		Length		No	Width		Length	
			Mean	S D	Mean	S D		Mean	S D	Mean	S D
1-3	♀	16	31	3	39	4.5	6	36	4	50	7
1-3	♂	27	31	2.5	40	5.4	5	38	3.8	46	7
3-7	♀	17	34	2.4	39	4.8	13	43	4.6	51	6.4
3-7	♂	57	34	2.4	43	3.7	5	44	3	51	3.3
7+		23	35	2.8	44	3.5	11	46	5	52	4.8
7+	♂	40	35	3.1	44	3.5	18	46	3.4	54	4.8

The measurements for the largest width of the foramen magnum were made from a 40 half axial view in supine position made on the Schonander skull unit (the magnification being about 25). The measurements for the length of the foramen magnum basion opisthion were made from special lateral films of the foramen magnum with a focus object distance of 75 cm and object film distance of 15 cm resulting in a magnification of 20.

expense (Fig 7 a) at the same time foreshortening the internal meatus (Fig 7 b). This sulcus is not produced by a dilated cistern or by an enlarged sinus but by pressure of the superior and anterior edge of the cerebellum which was found squeezed and lodged into the sulcus.

The third abnormality seen in 41 % was a flatness of the floor of the posterior fossa with a shallow posterior fossa and low position of the union (Fig 8).

These three abnormalities are summarized in Fig 9. The broken line shows that a triad of foramen magnum enlargement, flat floor of the posterior fossa and scalloping of the postero-medial aspects of the petrous bones was present in only 39 % of our cases. Complete normality was observed in 25 % while 36 % showed an incomplete triad.

Table 2 combines different skull types with the spinal defects which allows the following observations. A normal skull was not found with spinal defects above the lumbar area. The triad of skull abnormalities described here was seen in all categories of spinal defects except for the isolated sacral lesion. The incomplete triad was seen without special preference while an enlarged skull was a quite constant finding.

In none of these patients was evidence obtained of platybasia or basilar impression. In none was occipitalization of C1 seen and in only two was a

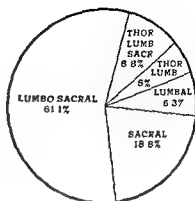


Fig. 2. Distribution of spinal defects in the 80 cases.

fusion of the bodies of C2 and C3 detected. In 20 %, however, a variable degree of displacement of the odontoid process, posteriorly to the line of THIEBAUT, was discovered (Fig. 10). Simultaneously, there was an enlarged foramen magnum. However, no luxation in the atlanto odontoid joint could be demonstrated. This displacement was present in the neutral position as well as in flexion and extension. It is probably relative and due to the widening of the foramen magnum anteriorly.

Literature

From the beginning we realized that these skull abnormalities stood in close relation to associated intracranial abnormalities. The abnormality most likely to be present was thought to be the Arnold Chiari deformity of the hindbrain, so often reported in association with meningocele.

In perusing the literature it was realized that many intracranial abnormalities in association with meningocele, other than the Arnold Chiari deformity, have been described. Reported are deformities and/or defects of the midbrain and aqueduct of Sylvius (6, 13, 16, 37, 39, 47, 51, 53), diencephalon (6, 20), corpus callosum (16), falx (6, 13), great sinus (6) and vein of Galen (6). The tentorium was found with shallow leaves and low peripheral attachment (6, 13). Hypoplasia or aplasia of the tentorium are recorded, while the incisura was invariably large. The transverse sinus was found displaced inferiorly, running near the rims of the foramen magnum (13). Glial nests are seen in the meninges (11), and heterotopic nodules in the ventricles are reported (6, 11, 16, 23).

The vertebral artery was seen lying in a deep groove of the pons (39, 51), and furthermore microgyria of cerebrum and cerebellum were seen, the latter being often small and asymmetrical (6, 8, 9, 13, 16, 50, 51, 53). The superior

Table 2

Distribution and frequency of the spinal defects in 80 patients with spina bifida in relation to their skull findings

Group	Skull Particulars	No	Spine				
			Thoraco-lumbar	Thoraco-lumbo-sacral	Lumbo-sacral	Lumbar	Sacral
1	Normal	20	—	—	11	1	8
2	Large foramen magnum Ossa petrosa scalloping Flat posterior fossa Enlarged skull	31	3	4	21	3	—
3	Large foramen magnum Ossa petrosa scalloping Large skull	17	—	2	11	—	4
4	Large foramen magnum Ossa petrosa scalloping	2	—	—	—	—	—
5	Same as sub 2 but without large skull	1	1	—	—	—	—
6	Same as sub 2 but foramen magnum not enlarged	1	—	1	—	—	—
7	Only foramen magnum enlarged Incomplete skull investigation	6 2	— —	— —	3 1	1 —	2 1
	Total	80	4	7	49	5	15

vermis is often flat and lies in the tentorial incisura. This flattening is due to the herniation (and pressure) of the overlying occipital lobe (53). Hydrocephalus is uniformly reported and congestion of the cerebellum could be found which is limited to that part of the hindbrain below the level of the foramen magnum.

Bony abnormalities however were dealt with less frequently except for the lacunar skull or Luckenschädel also found pathologically by CHIARI (9) and according to KARSNER & REEVES (33) first reported in 1771. However, this Luckenschädel was not encountered in this material in patients 1 year of age and up. RUSSELL reports scalloping of the basiocciput. Flattening of the floor of the posterior fossa was noted by HURTEAU in an Arnold Chiari deformity. However HURTEAU's case was probably not a proven Arnold Chiari (see Discussion).

Scalloping of the postero-medial aspects of the petrous bones does not seem to have been recorded before.

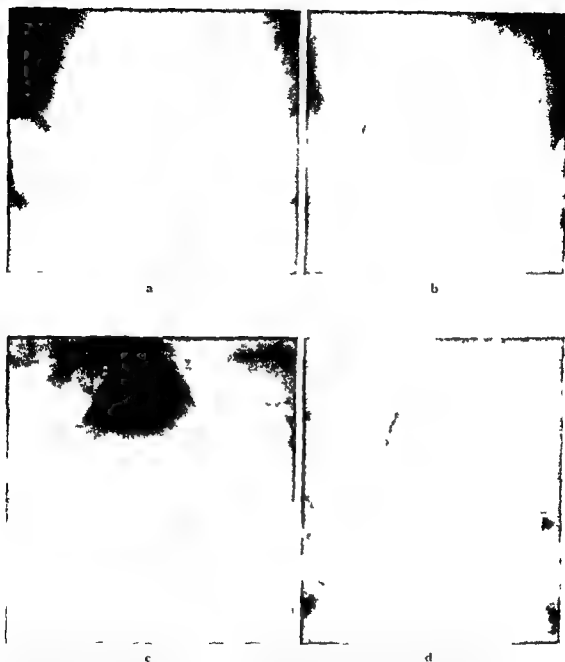


Fig. 3 Examples of shape and size of the foramen magnum in the Arnold Chiari skull. (a) A small incisura. (c) A more marked incisura at the level of the opisthion. The jugular tubercles are not well seen in (c) and (d) but should not be taken as a sign of posterior fossa tumor. (cf fig. 4)

Only in pathologic and neurologic publications has enlargement of the foramen magnum occasionally been mentioned and regarded as typical of the Arnold Chiari deformity (6, 9, 13, 41, 50, 58)



Fig 4 Foramen magnum of a normal child for comparison with fig 3 Figs 3 and 4 were taken with a ray direct on of 40 from above on the Schonander skull unit Age 8 to 10 years

Discussion

From the beginning we realized that these skull abnormalities stood in close relation to the spinal defects. The majority of reports associating spina bifida with other central nervous defects refer to the Arnold Chiari type of hindbrain deformity. RUSSELL and DANIEL & STRICH even state that it is clear that whenever a case of meningocele and particularly a meningomyelocele is seen the possibility of an associated Arnold Chiari deformity should be borne in mind. This deformity consists of a tail like elongation of the inferior vermis along the roof of the fourth ventricle extending downwards to a variable length over the spinal cord. Pons and medulla can be elongated as in the 4th ventricle and can be placed partially into the cervical canal as are the tonsils. CHIARI described this abnormality in 1891 and 1895. Since then numerous similar findings have been reported. The term Arnold Chiari deformity was coined by SCHWALBE & GREDIG in 1907.

In our material of 80 cases of spina bifida it is reasonable to expect the Arnold Chiari deformity in a high percentage. The above described skull abnormalities were thought to be sufficiently strong criteria in entertaining this diagnosis. The following supporting data are:

1. In 5 cases pneumocephalography and/or posterior fossa explorations have been carried out. In each hydrocephalus with a typical elongated 4th ventricle was demonstrated or the typical Arnold Chiari deformity was observed during exploration. Two had the complete triad, three had an incomplete triad (large foramen magnum and petrous bone scalloping).

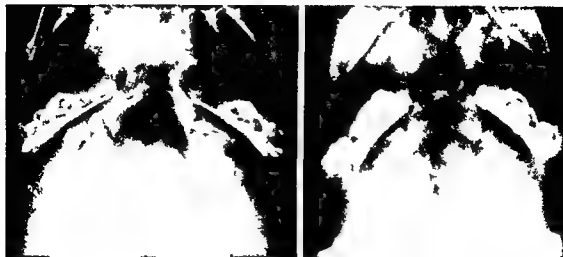
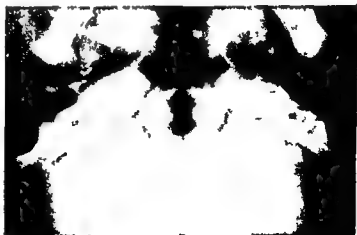


Fig 5 Three examples of bilateral scalloping of the posteromedial aspects of the petrous bones. Interpetrosal distance enlarged; meatus acusticus internus cut off medially. A waist is seen at the level where normally the petrosal bone has its largest width. Compare with fig. 6.



Fig 6 Aspects of petrosal bones in a normal skull.



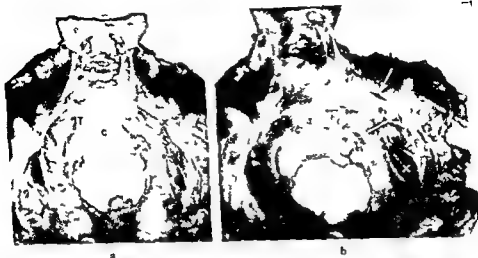


Fig 7 Specimen of posterior fossa of infant with meningocele and a proven Arnold Chiari deformity of the hindbrain a) Seen from above J — jugular tubercle T — superior petrosal ridge c — basioccipital s — deep sulcus running posteriorly to the petrosal bone and scalloping it inferiorly b) Specimen slightly rotated to show actual width of sulcus (l-i) — meatus acusticus m-a — entrance into foramen jugulare

2 Since this study was undertaken we have collected recent post mortem data in 10 infants with meningocele in which a routine radiologic skull investigation was carried out. Each of them demonstrated a large foramen magnum and the same typical scalloping of the petrous bones and in each of them the presence of the Arnold Chiari deformity of the hindbrain could be confirmed.

3 Pathologic and neurologic publications, (6 9 13 41 50 58) do refer to the enlarged foramen magnum in the Arnold Chiari deformity although often without concrete figures and seldom referring to the basion opisthion distance.

A large foramen magnum is not typical only for the Chiari type II or Arnold Chiari deformity. CHIARI himself described in his type III deformity of the hindbrain the presence of a large foramen magnum. This Chiari type III deformity is also known under the synonym Klippel Feil and anencephaly (9). In this type however severe cervical defects are encountered often with occipito cervical meningo-encephalocele and is easily recognized as such.

Flatness or straight floor of the posterior fossa which at the same time is shallow was seen in 41 %. Probably it is not pathognomonic for the Arnold Chiari deformity although HURTEAU reported this flattening as typical



Fig 8 Lateral view of the skull demonstrating the flat floor of a rather shallow posterior fossa

His patient, a 25 year old woman, however, did not have hydrocephalus and at posterior fossa exploration only severe tonsil herniation and adhesions were observed, findings of the typical triad which go with the Arnold Chiari deformity were not reported.

It is rather striking and significant that in none of our patients was there any evidence of platybasia, basilar impression or occipitalization of C1. Only twice was a fusion of the bodies of C2 and C3 noted. We do not think that these abnormalities are typical of the Arnold Chiari deformity, however, a rare exception will exist. In none of the classic pathologic publications (2, 6, 8, 9, 13, 21, 39, 51, 52, 53, 56) was this combination mentioned. Many published cases of Arnold Chiari deformity (3, 1, 10, 12, 25, 31, 38, 42, 43, 44, 46, 56, 59, 60) with congenital occipital bone abnormalities, platybasia, basilar impression and occipitalization of C1, are in fact not (proven) Arnold Chiari deformities with the possible exceptions of Cases 5 and 6 from List. The fact that in these cases the tonsils were found herniated is not evidence enough and not proof of the Chiari type II defect, but makes it more likely to classify them as possible Chiari type I deformities.

Also many publications (1, 5, 15, 22, 26, 35, 45, 57-59) claiming Arnold Chiari deformities without the presence of occipital bone and spine abnormalities, are in fact not proven Arnold Chiari, but possible Chiari type I deformities. Only PEACH's recent publication is probably the first proven Arnold Chiari deformity without spinal dysraphism. In fact, his paper is a welcome contribu-

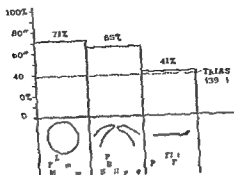


Fig 9 Skull abnormalities most frequently encountered in the material. Schematic drawing.

tion which happily refuted the myth about the Arnold Chiari deformity as caused by traction of a tethered cord and diastematomyelia.

We know from pathologic studies among others RUSSELL (51-53), that a wide range exists in the severity and degree of hindbrain deformities in meningocele patients. Grades of cerebellar hypoplasia are encountered. Probably the inconsistency of our radiologic findings depends on these varieties already documented by CHIARI in 1891 and 1895. The complete as well as the incomplete Arnold Chiari skulls were larger than normal with often evidence of a certain degree of arrested hydrocephalus. Detailed analysis of size and shape of these skulls was not helpful because of the variety of treatment (shunting procedures, repair of meningocele, conservative treatment) and age at which treatment was initiated. The upper cervical spine was not infrequently wider than normal. The patient's face was often rather without expression even in many with normal school progress. However, voluntary facial muscle movements were normal.

The number of normal skulls was rather large in this series. We know that spina bifida does occur without the Arnold Chiari deformity but the percentage here is rather high. Probably this may be properly explained by the fact that our group of patients is a self-selected group out of an original material of over 1500 cases of spina bifida of which the fittest survived.

With the progress in surgical procedures and chemotherapy, more and more of these patients are expected to survive and reach adolescence. Skull films made in these patients can inform us about possible problems and complications to be expected. We lost 4 patients rather suddenly, unfortunately without post mortem studies we are still in the dark as to whether their sus-



Fig. II Lateral view of the skull demonstrating the flat floor of a rather shallow posterior fossa

His patient, a 25 year old woman, however, did not have hydrocephalus and at posterior fossa exploration only severe tonsil herniation and adhesions were observed, findings of the typical kind which go with the Arnold Chiari deformity were not reported.

It is rather striking and significant that in none of our patients was there any evidence of platybasia, basilar impression or occipitalization of C1. Only twice was a fusion of the bodies of C2 and C3 noted. We do not think that these abnormalities are typical of the Arnold Chiari deformity, however a rare exception will exist. In none of the classic pathologic publications (2, 6, 8, 9, 13, 24, 39, 51, 52, 53, 56) was this combination mentioned. Many published cases of Arnold Chiari deformity (3, 1, 10, 12, 25, 34, 38, 42, 43, 44, 46, 56, 59, 60) with congenital occipital bone abnormalities platybasia, basilar impression and occipitalization of C1, are in fact not (proven) Arnold Chiari deformities, with the possible exceptions of Cases 5 and 6 from List. The fact that in these cases the tonsils were found herniated is not evidence enough and not proof of the Chiari type II defect, but makes it more likely to classify them as possible Chiari type I deformities.

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ing of the postero medial aspects of the petrous bones which was produced by a sulcus in this area. In this sulcus was lodged the supero anterior edge of the cerebellum. This scalloping was always seen in the presence of a large foramen magnum. Post mortem findings of the Arnold Chiari type of hindbrain deformity in a group of infants with meningocele and with the same findings as described above support the idea that the presence of the Arnold Chiari or Chiari type II deformity of the hindbrain can be suspected if such skull abnormalities are present.

SUMMARY

The radiologic features in patients with spina bifida are described. These are enlargement of the foramen magnum and scalloping of the postero-medial inferior aspects of the ossa petrosa. Flatness of the floor of the posterior fossa was also seen though less frequently. These findings some of them new are presented as evidence for the presence of Arnold Chiari deformity of the hindbrain. Because of this it was suggested that skulls with this triad of abnormalities be named the Arnold Chiari or Chiari type II skulls.

ZUSAMMENFASSUNG

Die radiologischen Zeichen in Patienten mit spina bifida werden beschrieben. Diese sind Vergrößerung des Foramen occipitale magnum und Unregelmässigkeiten im post medial inferioren Aussehen der Ossa petrosa. Platttheit des Bodens der hinteren Fossa war auch zu sehen obwohl weniger häufig. Diese Befunde von denen manche neu sind werden als Beweis für eine Arnold Chiari Missbildung des Rautenhirns gedeutet. Dieses berücksichtigend schlagen die Verfasser vor dass Schädel mit diesen Abnormalitäten Arnold Chiari oder Chiari Typ II Schädel genannt werden.

RÉSUMÉ

Description des signes radiologiques chez les malades atteints de spina bifida. Il y a agrandissement du trou occipital et irrégularités des parties postéro-internes et inférieures des rochers. On a constaté aussi mais moins souvent l'aplatissement du plancher de la fosse postérieure. Ces signes dont certains sont nouveaux sont présentés comme la preuve qu'il y a une malformation d'Arnold Chiari de la partie postérieure de l'encéphale. C'est pourquoi les auteurs proposent d'appeler crânes d'Arnold Chiari ou crânes de Chiari type II les crânes qui présentent cette triade d'anomalies.

REFERENCES

1. ARING C. B. Cerebellar syndrome in an adult with malformation of the cerebellum and brain stem (Arnold Chiari deformity) with a note on the occurrence of Torpedoes in the cerebellum. *J. Neurol. Neurosurg. Psychiat.* 1 (1938) 100.
2. ARNOLD J. Myelocyste. Transposition von Gewebskernen und Sympodie. *Beitr. path. anat.* 16 (1894) 1.



Fig. 10 Odontoid process seen protruding posteriorly to the line of Thiebaux

pected hydrocephalus and hindbrain deformity had contributed to their death.

The Arnold Chiari deformity might be suspected from skull films also in those cases in which no spine deformities are present. Furthermore, in the differential diagnosis of hydrocephalus, it is good to note that the skull abnormalities described and introduced here are not seen in Dandy Walker, hydrocephalus due to cisternal block or posterior fossa tumors.

Conclusion

In a routine skull investigation of neurogenic bladder patients, on the basis of a spinal bifida, it was found that certain deformities of the chondrocranium appeared in a high frequency. These skull abnormalities are 1) enlargement of the foramen magnum, 2) scalloping of the postero-medial inferior aspects of the petrous bones and 3) flat floor of the posterior fossa.

Evidence from the literature was collected which favours the view that an enlarged foramen magnum is present in the Arnold Chiari deformity of the hindbrain. Although such enlargement is also seen in the Chiari type III deformity, the latter can easily be differentiated from the Chiari II deformity.

A skull abnormality which appeared to be totally new was a peculiar scallop

- 24 GREENFIELD J G BLACKWOOD W McMENEMEY W H MEYER A and NORMAN R M
Neuropathology Edward Arnold (Publ) Ltd London
- 25 GROS C ROILEN A VLAHOVITCH II et ARTIGALA MIJOVIC Problemes diagnostiques
complexes posés par la maladie d Arnold Chiari Montpellier med 3 (1960) 231
- 26 HURTEAU E F Arnold Chiari malformation J Neurosurg 7 (1950) 282
- 27 INGRAHAM F D and SWAN H Spina bifida and cranium bifidum encephalocele A
survey of 546 cases New Engl J Med 228 (1943) 559
- 28 — and HAMLIN H Spina bifida and cranium bifidum Surgical treatment New Engl
Med J 228 (1943) 631
- 29 — and MATSON D D Neurosurgery of infancy and childhood Thomas Springfield Ill
1954
- 30 — and SCOTT H W Spina bifida and cranium bifidum The Arnold Chiari malforma
tion New Engl J Med 229 (1943) 108
- 31 JACOBS E B LANDING B H and THOMAS W Vermicomelia Amer J Path 39 (1961)
345
- 32 KAPSENBERG J G and VAN LOOKERV CAMPAONE J A Spina bifida combined with
diastematomyely anomaly of Chiari and hydrocephaly case Acta anat 7 (1949)
366
- 33 KARSNER R G and REEVES D L Lacunar skull (Luckenschadel) of the newborn
Amer J Roentgenol 57 (1947) 321
- 34 KISSEL P ARNOLD G TRIDON P et SCHMITT J Malformation rare de la charniere
occipito-vertébrale associée à une anomalie d Arnold Chiari et à un raccourcissement
congénital des deux humérus Rev neurol (1960) 109
- 35 KRAYENBUHL H Chronic internal hydrocephalus due to abnormality of cerebellum related
to Arnold Chiari deformity Schweiz med Wschr 71 (1941) 414
- 36 LAURENCE K M The natural history of Spina bifida cystica Proc roy Soc Med 53
(1960) 1055
- 37 LAVA S EYAL Z and SCHWARTZ A Neurological and surgical experiences in Arnold
Chiari malformation Acta neurochir (Wien) (1960) 532
- 38 LEVY J R WYCK H T and YOUNG B R Roentgen diagnosis of herniation of brain
into spinal canal (Arnold Chiari deformity) by pantopaque myelography Radiology
54 (1955) 591
- 39 LICHTENSTEIN B W Atresia and stenosis of the aqueduct of Sylvius J Neuropath exp
Neurol 18 (1959) 3
- 40 LICHTENSTEIN W B Spinal Dysraphism Spina bifida and myelodysplasia Arch Neurol
Psychiat 44 (1940) 792
- 41 LITT C F Neurologic syndromes accompanying developmental anomalies of occipital
bone atlas and axis Arch Neurol Psychiat 45 (1941) 578
- 42 LOCHNER L E BAKER G S and MILDNER D W Arnold Chiari malformation in adults
Report of a case Proc Mayo Clin 32 (1957) 632
- 43 MALIS L I COHEN I and GROSS S W Arnold Chiari malformation Arch Surg 63
1951 783
- 44 MARKS J H and LIVINGSTONE K E The cervical subarachnoid space with particular
reference to syringomyelia and the Arnold Chiari deformity Radiology 52 (1949)
67
- 45 MCCONNELL A A and PARKER H L Deformity of hind brain associated with internal
hydrocephalus its relation to Arnold-Chiari malformation Brain 61 (1938) 415

- 3 BARTSTRA H. K. Malformatie van Arnold Chiari en Platybasia. *Folia psychiat neerl* 57 (1954) 104
- 4 BLOOM W. H. Arnold Chiari malformation: report of a case. *Arch Neurol Psychiat* 66 (1951) 228
- 5 BUGY P. and LICHTENSTEIN B. W. Arnold Chiari deformity in adult without obvious cause. *J Neurosurg* 11 (1945) 245
- 6 CAMERON A. H. The Arnold Chiari and other neuro-anatomical malformations associated with spina bifida. *J Path Bact* 73 (1957) 195
- 7 — Malformation of the neuro spinal axis, urogenital tract and foregut in spina bifida attributable to disturbances of the blastopore. *J Path Bact* 73 (1957) 213
- 8 CHIARI H. Über Veränderungen des Kleinhirns des Pons und der medulla oblongata in Folge von congenitaler Hydrocephalie des Grosshirns. *Denkschr Akademie d Wissensch* 63 (1895) 71
- 9 — Über Veränderungen des Kleinhirns in Folge von Hydrocephalie des Grosshirns. *Dtsch med Wschr* 42 (1891), 1172
- 10 COCAN D. C. and LAWRENCE J. B. Platybasia and the Arnold Chiari malformation. *A M A Arch Ophthalm* 52 (1954) 13
- 11 COOPER I. S. and AFROHAN J. W. Heterotopic glial nests in subarachnoid space: Histopathologic characteristics, mode of origin and relation to meningeal gliomas. *J Neuropath exp Neurol* 10 (1951) 16
- 12 COSSA P., DUPLAY M., MARTIN J., AUBERT D. et HENRI M. Malformation D Arnold Chiari. Impression basilaire invagination basilaire platybasie convexobasie aplasie de la lame basilaire. A propos de deux observations. *Rev neurol* 85 (1931) 392
- 13 DANIEL P. M. and STRICH J. Some observations on the congenital deformity of the central nervous system known as the Arnold Chiari malformation. *J Neuropath exp Neurol* 17 (1958) 255
- 14 DORAN P. A. and GUTHRIE G. A. W. Studies in spina bifida. Part IV. The frequency and extent of paralysis. *J Neurol Neurosurg Psychiat* 26 (1963) 449
- 15 EPSTEIN B. S. Pantographic myelography in the diagnosis of the Arnold Chiari malformation without concomitant skeletal or central nervous system defects. *Amer J Roentgenol* 59 (1948) 359
- 16 FLICIN I. Arnold Chiari malformation with associated analogous malformation of the midbrain. *Neurology* 6 (1956) 22
- 17 GARDNER W. J. Anatomic features common to the Arnold Chiari and the Dandy Walker malformations suggest a common origin. *Cleveland Clin Found* 26 (1959) 206
- 18 — Anatomic anomalies common to myelomeningocele of infancy and syringomyelia of adulthood suggest a common origin. *Cleveland Clin Quart* 26 (1959) 118
- 19 — Rupture of the neural tube. *Arch Neurol* 4 (1961) 1
- 20 — Myelomeningocele: the result of rupture of the embryonic neural tube. *Cleveland Clin Quart* 27 (1960) 88
- 21 — and COLLIS J. S. Klippel Feil syndrome (syringomyelia diastematomyelia and myelomeningocele) — one disease? *Arch Surg* 83 (1961) 638
- 22 — and GOODALL R. J. Surgical treatment of the Arnold Chiari malformation in adults. *J Neurosurg* 7 (1950) 199
- 23 — ABDULLAH A. F. and MCCORMACK L. J. Varying expressions of embryonal atresia of fourth ventricle in adults. Arnold Chiari malformation. Dandy Walker syndrome. arachnoid cyst of cerebellum and syringomyelia. *J Neurosurg* 14 (1957) 591

- 24 GREENFIELD J G BLACKWOOD W McMENEY W H MEYER A and NORMAN R M
Neuropathology Edward Arnold (Publ) Ltd London
- 25 GROS C ROUGEN A VLAHOVITCH B et ARTIGALA MIJOVIC Problèmes diagnostiques
complexes posés par la maladie d'Arnold Chiari Montpellier med 3 (1960) 231
- 26 HURTEAU E F Arnold Chiari malformation J Neurosurg 7 (1950) 282
- 27 INGRAHAM F D and SIVAK H Spina bifida and cranium bifidum encephalocele A
survey of 546 cases New Engl J Med 228 (1943) 559
- 28 — and HAMLIN H Spina bifida and cranium bifidum Surgical treatment New Engl
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- 29 — and MATSON D B Neurosurgery of infancy and childhood Thomas Springfield Ill
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- 30 — and SCOTT H W Spina bifida and cranium bifidum The Arnold Chiari malforma-
tion New Engl J Med 229 (1943) 108
- 31 JACOBS E B LANDING B H and THOMAS W Vernicomelia Amer J Path 39 (1961)
345
- 32 KAPENBERG J G and VAN LOOKERV CAMPAGNE J A Spina bifida combined with
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366
- 33 KARSNER R G and REEVES D L Lacunar skull (Luckenschadel) of the newborn
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- 34 KAMEL F ARNOLD G TRIDON P et SCHMITT J Malformation rare de la charniere
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- 35 KRAYENBUHL H Chronic internal hydrocephalus due to abnormality of cerebellum related
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- 36 LAURENCE K M The natural history of Spina bifida cystica Proc roy Soc Med 53
(1960) 1055
- 37 LAVY S EYAL Z and SCHWARTZ A Neurological and surgical experiences in Arnold
Chiari malformation Acta neurochir (Wien) (1960) 519
- 38 LEWIS J E WYCKS H T and VOLAG H R Roentgen diagnosis of herniation of brain
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54 (1950) 591
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- 41 LEST C F Neurologic syndromes accompanying developmental anomalies of occipital
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- 43 MALIS L I COHEN I and GROSS S W Arnold Chiari malformation Arch Surg 63
(1951) 783
- 44 MARKS J H and LIVINGSTONE K E The cervical subarachnoid space with particular
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- 45 MCCONNELL A A and PARKER H L Deformity of hind brain associated with internal
hydrocephalus its relation to Arnold Chiari malformation Brain 61 (1938) 415

- 46 MULLAN S. and RAIMONDI A. J. Respiratory hazards of the surgical treatment of the Arnold Chiari malformation. *J. Neurosurg.* 19 (1962), 675.
- 47 OGRYZLO M. A. The Arnold Chiari malformation. *Arch. Neurol. Psychiat.* 48 (1942), 30.
- 48 PEACH H. Arnold Chiari Malformation with normal spine. *A.M.A. Arch. Neurol.* 10 (1964), 497.
- 49 PENFIELD W. and COBURN D. F. Arnold Chiari malformation and its operative treatment. *Arch. Neurol. Psychiat.* 40 (1938), 328.
- 50 RICARD A. et GIRARD P. F. Différentiel d'Arnold Chiari. *Rev. neurol.* 81 (1949), 332.
- 51 RUSSELL D. S. and DONALD C. The mechanism of internal hydrocephalus in spina bifida. *Brain* 58 (1935), 203.
- 52 — Hydrocephalus in neurology and psychiatry in childhood. *Res. Publ. Ass. nerv. ment. Dis.* 34 (1954), 160.
- 53 — Observations on the pathology of hydrocephalus. *Spec. Rep. Ser. med. Res. Coun.* No. 265 (1949).
- 54 SANDERSON U. Le syndrome d'Arnold Chiari. *Rev. neurol.* 93 (1955), 529.
- 55 SCHMID W. Ein Fall von Hypoplasie und einseitiger Assimilation des Atlas mit Platibasis basilärer Impression und Arnold Chiari'scher Anomalie. *Schweiz. Arch. Neurol.* 88 (1961), 147.
- 56 SCHWALBE F. und GREDER M. Über Entwicklungsstörungen des Kleinhirns, Hirnstammes und Halsmarks bei spina bifida. *Beitr. path. Anat.* 40 (1907), 132.
- 57 SWANSON H. S. and FINCHER E. F. Arnold Chiari deformity without bony anomalies. *J. Neurosurg.* 6 (1949), 314.
- 58 THIENAUT F. Le diagnostic radiologique des malformations cérébello-bulbaires décrites par Chiari. *Roentgen Europ.* 6—7 (1962), 209.
- 59 VERBIST H. The Arnold Chiari malformation in adults without concomitant anomalies of the skull or the vertebral column. *Folia psychiat. neerl.* 56 (1953), 544.
- 60 — The Arnold Chiari malformation. *J. Neurol. Neurosurg. Psychiat.* 16 (1953), 227.

ROENTGENOLOGY OF HOLOPROSENCEPHALY (Arhinencephaly)

by

G J HURLANDER W DEWEYER J A CAMPBELL and H TAYBI

The classification of malformations recognized under the heading arhinencephaly has recently been revised. A generic term holoprosencephaly has been suggested (ref 5). This communication will outline the developmental anatomy of holoprosencephaly and the accompanying craniofacial malformations. The intimate relationship between prosencephalic and median facial development provides a reliable means of predicting the more severe degrees of brain malformation on the basis of the clinical and roentgen features of the facial configuration (ref 7).

Developmental anatomy Fig. 1 depicts the three primary and five secondary brain vesicles that ultimately develop into the adult brain. In the severe form of holoprosencephaly (alobar) the prosencephalon remains as a holosphere with a single ventricular cavity. This holosphere is covered by gyri with cerebral cortex and corresponds to the posterior frontal parietal occipital and temporal lobes of the normal brain. The anterior portions of the frontal lobes, the frontal poles, do not develop. A fluid containing dorsal sac found posterior to the prosencephalon probably represents the evaginated roof of the prosen-

Table

Face	Brain	Cranium	Orbit
Cyclopia	Alobar holoprosencephaly: small monoventricular undivided cerebrum; roof of cerebral ventricle a thin membrane which may be ballooned dorsally forming a dorsal sac; absent olfactory bulbs and tracts; corpus callosum absent or rudimentary; unseparated thalamus; Hippocampi remain in dorsal position	Microcephaly	Single
Ethmocephaly		Microcephaly	Two but extreme hypotelorism
Cebacephaly		Microcephaly	Hypotelorism
With median cleft lip		Microcephaly and sometimes trigonocephaly	Hypotelorism
Too few facial descriptions to establish a pattern	Semilobar holoprosencephaly: small brain; cerebral lobes present but rudimentary; interhemispheric fissure lacking; cerebral neocortex in continuity across midline; olfactory bulbs and tracts usually but not always absent; corpus callosum rudimentary	Microcephaly	Hypotelorism or hypertelorism (?)
Philtrum premaxilla large (brain type A; lobar holoprosencephaly)	<i>Lobar holoprosencephaly</i> Type A — With midline continuity of frontal neocortex Type B — With complete separation of neocortex across the midline Well formed lobes which may be of normal size; interhemispheric fissure present but may be interrupted anteriorly if neocortex in continuity across midline; olfactory bulbs and tracts may or may not be present; corpus callosum may be absent, hypoplastic or normal; a dorsal sac may be present communicating with the third ventricle and extending upwards between the hemispheres	Microcephaly and sometimes trigonocephaly	Hypotelorism
May or may not have obvious malformations (brain type A or B; lobar holoprosencephaly)		May have trigonocephaly	Hypotelorism, normal or hypertelorism (?)

Table (cont.)

Nose	Proboscis	Upper lip	Primary palate	Maxillae and secondary palate**	Middle facial bones
Absent	Present above orbit	Uncleft	Absent	Underdeveloped and fused or in direct contact	Absent
Absent	Present	Uncleft	Absent	Underdeveloped and fused or in direct contact	Absent
Flat and rudimentary with a single median nostril	Absent	Uncleft	Absent	Fused or in direct contact	Absent
Flat	Absent	Median cleft	Often absent	Usually with cleft secondary palate	Absent
	Absent				
Nasal bridge present but hypoplastic	Absent	Bilateral lateral cleft	Present but hypoplastic	Cleft secondary palate	Present but rudimentary
Nasal bridge present and well formed	Absent	May have unilateral or bilateral cleft	Hypoplastic or normal	Secondary palate may or may not be cleft	Present

Primary palate = anterior to incisive foramen. It is a lip structure developmentally (ref. 11).

Secondary palate = posterior to incisive foramen. It develops bilaterally from the palatine processes of the maxillary bones after the oral

Table

Face	Brain	Cranium	Orbit
Cyclopi	Alobar holoprosencephaly: small monoventricular undivided cerebrum; roof of cerebral ventricle a thin membrane which may be ballooned dorsally forming a dorsal sac; absent olfactory bulbs and tracts; corpus callosum absent or rudimentary; unseparated thalami; Hippocampus remains in dorsal position.	Microcephaly	Single
Ichthyocephaly		Microcephaly	Two but extreme hypotelorism
Cebocephaly		Microcephaly	Hypotelorism
With median cleft lip		Microcephaly and sometimes trigonocephaly	Hypotelorism
Too few facial descriptions to establish a pattern	Semilobar holoprosencephaly: small brain; cerebral lobes present but rudimentary; interhemispheric fissure lacking; cerebral neocortex in continuity across midline; olfactory bulbs and tracts usually but not always absent; corpus callosum rudimentary.	Microcephaly	Hypotelorism or hypertelorism (?)
<p>Thalrus premaxilla and large (brain type A: lobar holoprosencephaly)</p> <p>May or may not have obvious malformations (brain type A or B: lobar holoprosencephaly)</p>	<p><i>Lobar holoprosencephaly</i></p> <p>Type A - With midline continuity of frontal neocortex</p> <p>Type B - With complete separation of neocortex across the midline</p> <p>Well formed lobes which may be of normal size; interhemispheric fissure present but may be interrupted anteriorly if neocortex in continuity across midline; olfactory bulbs and tracts may or may not be present; corpus callosum may be absent, hypoplastic or normal; a dorsal sac may be present communicating with the third ventricle and extending upwards between the hemispheres</p>	<p>Microcephaly and sometimes trigonocephaly</p> <p>May have trigonocephaly</p>	<p>Hypotelorism</p> <p>Hypotelorism normal or hypertelorism (?)</p>



Fig. 1. Case 1. Microcephaly, hypotelorism and median cleft lip facial type. Keel shaped forehead and supra-orbital depression of trigonocephaly. (Reproduced by permission from Confinia Neurologica.)

fissure are well formed but one or more anomalies reflect the tendency for incomplete prosencephalic development. These anomalies include among others midline continuity of the frontal neocortex, agenesis of the olfactory bulbs and tracts, broad communication of the lateral ventricles and agenesis or hypoplasia of the corpus callosum with or without a dorsal sac. Since defects in development of the corpus callosum may result from a variety of mechanisms not all cases of this anomaly are related to holoprosencephaly.

Brain and craniofacial relationships Embryologically the spinal cord and brain stem up to the caudal limit of the prosencephalon are induced by notochordal mesoderm. The prosencephalon is induced by the prechordal mesoderm between it and the foregut (ref. 9). This prechordal mesoderm not only induces prosencephalic development but also is the anlage of the median facial structures which are the ethmoid including the crista galli, nasal and vomer bones as well as the nasal septum. Also included in the median facial structures are the components of the upper oral arch of naso-medial (medial) origin which are called the intermaxillary segment. The intermaxillary segment is comprised of three parts: 1) a labial component which forms the philtrum of the upper lip; 2) an upper jaw component, the premaxilla which normally carries four incisor teeth; and 3) a palatal component which forms the triangular primary

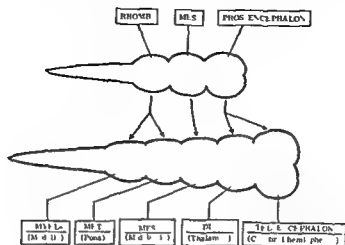


Fig. 1 The three primary and five secondary brain vesicles that ultimately develop into the normal brain

cephalic monoventricle. In addition, the cerebrum arrested at this stage of development contains unseparated thalamus. The olfactory bulbs and tracts are absent because they fail to differentiate, and the hippocampus remains in dorsal position. Absence of the olfactory bulbs and tracts led KUNDURAT to call the malformation 'rhinencephaly'. Several other tectogenic transformations fail to occur in this primitive brain (ref. 5).

Since the arrest in cerebral development can occur at successively later stages of embryogenesis a spectrum of related but less severe malformations is encountered. KUNDURAT and later authors have considered this spectrum to extend from cyclopia to agenesis of the corpus callosum: 1) cyclopia, 2) ethmocephaly, 3) cebocephaly, 4) with median cleft lip, 5) with bilateral lateral cleft lip, 6) with trigonocephaly, and 7) with agenesis of the corpus callosum (ref. 2). Although these groups have been classed under the heading 'rhinencephaly', the term is unsatisfactory because undue stress is placed on only one of the many cerebral defects, and in fact some brains belonging to this teratologic series have olfactory bulbs and tracts (ref. 5). Holoprosencephaly was suggested because it connotes an arrest in cleavage of the prosencephalon into cerebral hemispheres, and emphasizes the holistic, primitive nature of the undivided prosencephalon (ref. 5).

Classification. In lobar holoprosencephaly the most severe form of this brain malformation, no interhemispheric fissure and no lobes are present. It is found in cyclopia, ethmocephaly, cebocephaly, and median cleft lip (see Table). In semilobar holoprosencephaly lobes are recognized but no interhemispheric fissure is present. In lobar holoprosencephaly, lobes and an interhemispheric

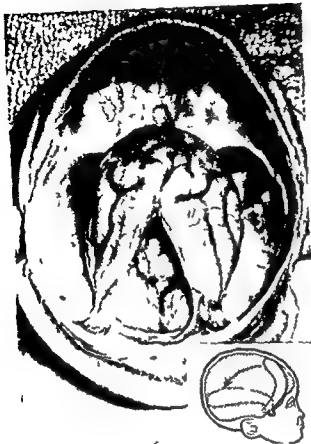


Fig 4 Case 1 Alobar holoprosencephalon as seen at post mortem with superior part of calvaria removed. Thin meningeal membrane forming the dorsal sac has also been removed. Large fluid filled spaces were present in front of and behind the cerebral substance. Diagram at lower right illustrates the variable position of the holotelencephalon within the skull. Fluid posterior to the holotelencephalon is contained in the dorsal sac which is completely separate from the space in front. (Reproduced by permission from Archives of Neurology.)

not developed sufficiently to separate the occipital lobes. The literature contains incomplete descriptions of the facial patterns in this group of cerebral malformations. Thus the reliable predictability of the brain malformation by the craniofacial configuration in alobar holoprosencephaly has not been firmly established with semilobar holoprosencephaly. The facial features noted in the few completely reported cases included broad flat nose, and hypotelorism with or without trigonocephaly.

The brains with lobar holoprosencephaly have well formed lobes and may be of normal size. An interhemispheric fissure is present but may be interrupted anteriorly if the frontal neocortex is in continuity across the midline. Many patients in this group will not have obvious facial malformations. Those who do may have hypotelorism with or without trigonocephaly, cleft lip or cleft palate.

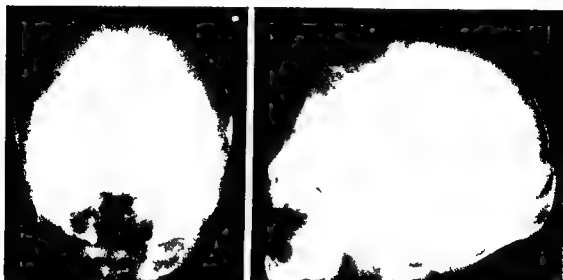


Fig. 3 Case 1 Frontal and lateral skull roentgenograms indicate microcephaly and orbital hypotelorism. Longest diameters of the orbits are convergent superomedially. Nasal and median facial bones are absent. Metopic suture is open (postmortem confirmation).

palate. The intermaxillary segment is continuous with the rostral portion of the nasal septum which is also of medial origin. The major portion of the definitive palate is formed by the palatine shelves which arise laterally from the maxillary swellings. These shelves fuse in the midline and form the secondary palate. Anteriorly they fuse with the triangular primary palate. The midline boundary between the primary and secondary palates is the incisive foramen (ref. 8). Interference with prechordal mesoderm, therefore, can cause defects in the median facial bones and intermaxillary segment as well as incomplete prosencephalic development. Disturbances in the inductive activity of the prechordal mesoderm might be caused by a number of factors including physical, toxic, metabolic or genetic disturbances all leading to similar craniofacial and brain malformations (ref. 5).

Craniofacial malformations are invariably present in alobar holoprosencephaly. Orbital hypotelorism is constant. Other anomalies which may appear in varying degrees and combinations are absence of the nasal septum, median and bilateral lateral clefts of the lip and primary palate, median cleft of the secondary palate, microphthalmia, anophthalmia, micrognathia, small frontal bones sometimes with trigonocephaly, and anurism with or without a proboscis. Patients with cyclopia, ethmocephaly, cebocephaly, and median cleft lip are included in this category of brain and facial malformations.

In semilobar holoprosencephaly the brain is small but cerebral lobes can be identified. The interhemispheric fissure may be suggested posteriorly but is

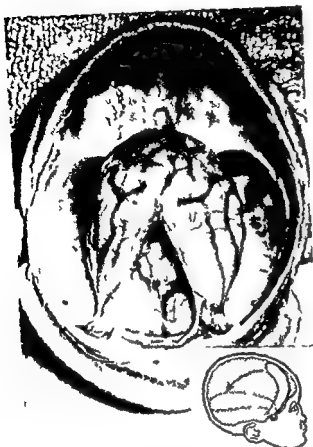


Fig 4 Case 1 Alobar holoprosencephalon as seen at post mortem with superior part of calvaria removed. Thin membrane forming the dorsal sac has also been removed. Large fluid filled spaces were present in front of and behind the cerebral substance. Diagram in lower right illustrates the variable position of the holotelenkephalon within the skull. Fluid posterior to the holotelenkephalon is contained in the dorsal sac which is completely separate from the space in front. (Reproduced by permission from Archives of Neurology.)

not developed sufficiently to separate the occipital lobes. The literature contains incomplete descriptions of the facial patterns in this group of cerebral malformations. Thus the reliable predictability of the brain malformation by the craniofacial configuration in alobar holoprosencephaly has not been firmly established with semilobar holoprosencephaly. The facial features noted in the few completely reported cases included broad flat nose and hypotelorism with or without trigonocephaly.

The brains with lobar holoprosencephaly have well formed lobes and may be of normal size. An interhemispheric fissure is present but may be interrupted anteriorly if the frontal neocortex is in continuity across the midline. Many patients in this group will not have obvious facial malformations. Those who do may have hypotelorism with or without trigonocephaly, cleft lip or cleft palate.



Fig. 2 Case 2 Frontal and lateral skull roentgenograms indicate orbital hypotelorism. Nasal and median facial bones are absent. Palatine processes of the maxillary bones are fused in the midline (arrow). Ethmoidal labyrinths are hypoplastic. Longest diameters of the orbits in the frontal view are vertically oriented and contour of superior margins is semicircular (half moon).

Case material Seventeen patients with holoprosencephaly have been studied at this institution in the recent past, and almost all have had complete post mortem examinations. Of this group, 10 had satisfactory roentgen evaluation of the skull or brain. Brief summaries of 6 of these 10 cases are presented below.

The behavior pattern has been quite similar in patients with the most severe form of this brain malformation, alobar holoprosencephaly (ref. 5). These patients were able to swallow, cry, and move their extremities and eyes but none showed any developmental progress. They all had numerous convulsions and poikilothermia. Even with a nutritionally adequate diet they failed to gain weight. None of this severely affected group survived infancy. Some patients with semilobar holoprosencephaly, however, will survive into childhood but will be mentally defective. Patients with lobar holoprosencephaly may reach adulthood and have normal mentality but many, if not most, will also be mentally defective.

Case reports

Case 1 S. O. was a 3 month old female with microcephaly (Fig. 2). The occipito frontal circumference was 27.5 cm. Physical examination revealed that her palpebral fissures had a distinct mongoloid tilt. She had orbital hypotelorism with clinical evidence of trigonocephaly which consists of a keel shaped forehead, orbital hypotelorism and supraciliary depression extending laterally to the temples. Because of a median cleft of her upper lip, gum, primary and secondary palates, the nasal and oral cavities communicated. The nasal septum was absent.



Fig 6 Case 3 Frontal and lateral skull roentgenograms indicate orbital hypotelorism. Nasal and median facial bones are absent. Nasogastric tube (arrow) rests on the palatine processes of the maxillary bones which form the secondary palate. Primary palate is absent in median cleft facial type.

Roentgenograms of the skull and face in frontal and lateral projections indicate absence of the nasal bones and extreme orbital hypotelorism (Fig 3). The vomer, perpendicular plate of the ethmoid, and the crista galli are absent. The cranial sutures including the metopic are open, and the maxillae and mandible are hypoplastic, the former to a more severe degree. The skull is small, particularly in the region of the anterior cranial fossa. The soft tissues of the shallow bony orbits protrude anteriorly. Two small frontal bones are present. At necropsy an alobar holoprosencephalon was found which was very small compared to the intracranial volume (Fig 4). It was attached to the region of the posterior fontanelle leaving fluid-filled spaces in front and behind. The space between the holotelencephalon and the cerebellum was occupied by the dorsal sac which was completely separate from the space in front. The metopic suture was open in its entirety.

Comment: It is evident from this case that closure of the metopic suture is not essential for the presence of trigonocephaly (ref 10).

Case 2: J A was a 3 week-old male with microcephaly (Fig 8). The occipito-frontal circumference was 31 cm. Physical examination indicated orbital hypotelorism with a median cleft lip and a united secondary palate. There was no trigonocephaly. A chromosome study indicated an apparently normal 46 chromosome karyotype. Roentgenograms in frontal and lateral projections indicate marked orbital hypotelorism (Fig 5). The cranial sutures are open. The nasal bridge is flat, and the soft tissues of the shallow bony orbits are protruding anteriorly. The nasal and vomer bones are absent, and the ethmoidal labyrinths are hypoplastic. The crista galli is not identified. The palatine processes of the maxillary bones are fused in the midline. The anterior cranial fossa and frontal bones are small. Necropsy disclosed an alobar holoprosencephalon.

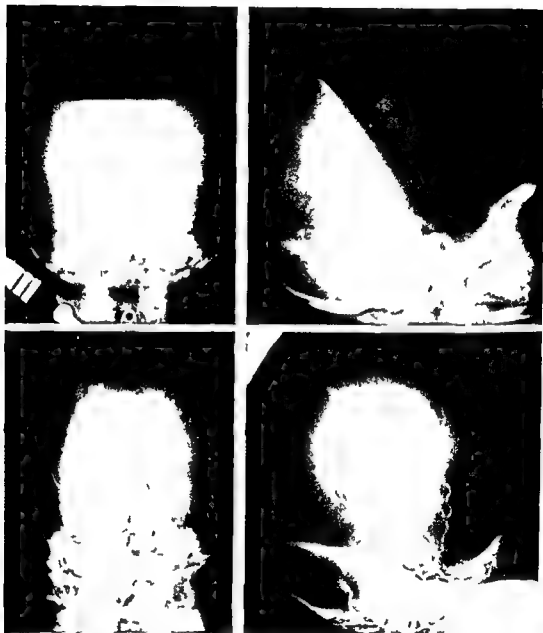


Fig. 7. Case 3. Ventriculograms in frontal and lateral projections (above) indicate large space interior to the cerebral mantle and the posterior dorsal sac. Carotid arteriograms (below). Arteries around brain substance are middle cerebral and possibly anterior cerebral arteries.

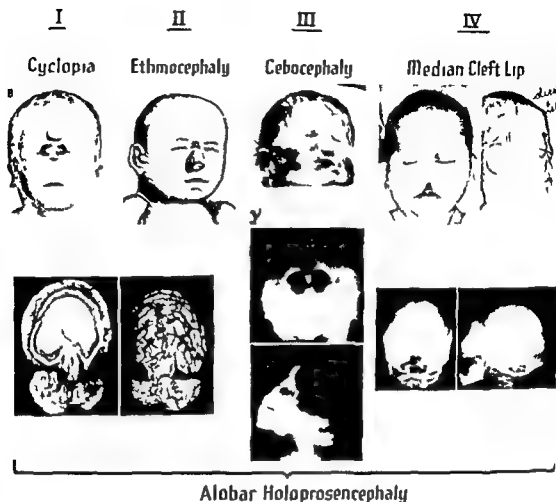
Case 3 A H was a 6-month-old female with an occipito-frontal circumference of 40.11 cm. Physical examination indicated orbital hypotelorism with a wide median cleft in the upper lip and gum. The secondary palate was intact and the orbital fissures had a mongoloid tilt. There was no trigonocephaly. The nasal septum was absent. Roentgenograms in frontal and lateral projections at one week of age indicate marked orbital hypotelorism (Fig. 6). The crista galli is not seen. The vomer and nasal bones as well as the perpendicular plate of the ethmoid bone are absent. The ethmoidal labyrinths are hypoplastic as are the maxillae. The palatine processes are fused in the midline. Orbital soft tissues protrude from the shallow bony orbits. The cranial sutures are open and two small frontal bones are present. The anterior cranial fossa is small. Ventriculography and arteriography performed at six months of age outline the cerebral mantle with the large dorsal sac posterior and above the tentorium cerebelli (Fig. 7). A large volume of fluid occupies the space anterior to the holoprosencephalon. Necropsy confirmed the presence of an alobar holoprosencephalon.

Comment: Cases 2 and 3 had the characteristic clinical features of median cleft lip. Although the median facial bones derived from prechordal mesoderm were defective, the secondary palate derived from fusion of the laterally originating palatine processes of the maxillary bones was intact. Apart from this interesting difference, the roentgen craniofacial configurations were almost identical in the first 3 patients.

Case 4 R was a white male infant who died 9 hours after delivery by caesarean section (Fig. 8). The occipito-frontal circumference was 27.5 cm. Physical examination indicated a rudimentary nose with a single median nostril. The lip was united in the midline but without a distinct philtrum. The only extracephalic malformations were single transverse palmar creases and undescended testicles. Chromosome studies from a skin culture disclosed an apparently normal 46 chromosome karyotype. Roentgenograms of the skull were done after removal of the brain and calvaria. There was marked orbital hypotelorism with a nasal space wedged between the orbits. The maxillae were hypoplastic and fused in the midline. The nasal bones were absent but hypoplastic osseous components (ethmoid) were present in the interorbital space. The brain malformation as seen at postmortem was that of alobar holoprosencephaly. No thoracic-abdominal visceral malformations were present.

Comment: Case 4 had typical cecocephalic facies. The roentgen craniofacial configuration was most interesting with a rudimentary nasal space wedged between two orbits which were very close together. The maxillae were fused in the midline and there was microcephaly. Cases 1 through 4 had strikingly similar brain malformations of alobar holoprosencephaly.

Case 5 M D was a 4-month-old female with macrocephaly. The occipito-frontal circumference was 38.5 cm. There was minimal clinical evidence of trigonocephaly. Physical examination indicated bilateral lateral clefts of the lip and primary palate with a median process (philtrum, premaxilla anlage or intermaxillary segment) extending from the region of the upper lip to the uvula. This median process continued upward as the nasal septum and could be seen extending to the posterior wall of the pharynx with which it united. Chromosome studies of the peripheral blood and skin from the median process disclosed an apparently normal 46 chromosome karyotype. During the latter months of life, a single incisor tooth erupted from the median process, establishing that a premaxilla anlage was present. Skull roentgenograms in frontal and lateral projections outline the median process and the unerupted single incisor (Fig. 8). The anterior cranial fossa is small as are the frontal bones. Nasal bones and a midline septum are visible. Postmortem study of the brain indicated lobar holoprosencephaly. type A (see Table).



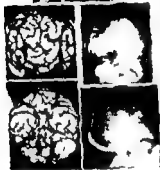
Alobar Holoprosencephaly

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Comment Case 5 had a more advanced degree of median facial development with nasal bones a nasal septum primitive philtrum and premaxilla (philtrum premaxilla anlage) with a single central incisor which was unerupted at the time of roentgen examination. Correspondingly there was a more advanced degree of brain development — lobar holoprosencephaly (type A see Table) (ref 7).

Case 6 SD was a 5 1/2 week old male with facial features suggestive of cebocephaly. The occipito frontal circumference was 28 cm. There was no clinical evidence of trigonocephaly. Physical examination revealed the upper lip to be united in the midline but without a distinct philtrum. There was a rudimentary nose and a single aperture to the nasal cavity. There was no cleft palate. The eyes were prominent. Frontal and lateral skull roentgenograms indicated microcephaly. The orbits were shallow and the protruding orbital soft tissues were seen in the lateral projection. The anterior cranial fossa was small. Small nasal bones were identified but the nasal bridge was flat. There was orbital hypotelorism. A midline osseous structure was seen in the frontal projection probably representing the perpendicular ethmoid.

V
Philtrum
premaxilla anlage



Lobar Holoprosencephaly

Fig 8 Composite clinical roentgen and neuroanatomic features of holoprosencephaly. Roentgenograms and anatomic specimens not necessarily those of patients depicted.

Brain in lower left is prototype of alobar holoprosencephaly. Cavity in dorsal view (left) represents single ventricle of the cerebrum. Corpora striata and thalami are uncleft in the midline. Ventral view (right) indicates absence of olfactory bulbs and tracts but all other cranial nerves intact.

Brain in column V is lobar holoprosencephaly type A. Interhemispheric fissure extends only to frontal lobes which are fused. Falx cerebri present posteriorly only. Absence of olfactory bulbs and tracts.

Case 2 Median cleft lip facial type is seen heading column IV. (Reproduced by permission from Archives of Neurology.)

Case 3 Cebocephaly is seen heading column III. (Reproduced by permission from Ann. Paediat. S. Karger Basel/New York.)

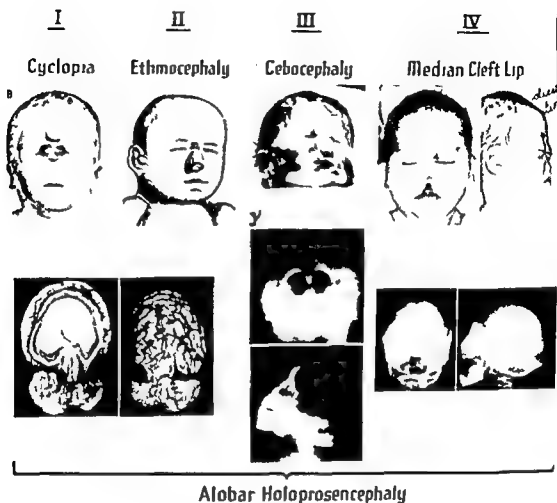
Case 5 Roentgenograms in column V indicate median process with solitary unerupted incisor seen in slightly oblique frontal projection. Nasal bones evident in the lateral projection.

plate and perhaps the vomer. The other portions of the ethmoid bone including the conchae were hypoplastic as were the maxillae. The cranial sutures were open. At postmortem this patient had lobar holoprosencephaly type A. No extracerebral anomalies were present.

Comment. Case 6 is interesting in that clinically the facial features suggested cebocephaly with a rudimentary nose and a single nasal aperture. There was no distinct philtrum present in the upper lip. The roentgen features of the face, however, indicated the presence of nasal and median facial bones suggesting a more advanced degree of median facial development than anticipated in true cebocephaly. The brain malformation, lobar holoprosencephaly type A, corresponded with the more advanced degree of facial bone development indicated by the roentgenograms.

Discussion

The majority of the seventeen cases of holoprosencephaly in this series were of the median cleft lip facial type. One case of cebocephaly was encountered



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Comment Case 5 had a more advanced degree of median facial development with nasal bones, a nasal septum, primitive philtrum and premaxilla (philtrum premaxillary enlargement) with a single central incisor which was unerupted at the time of roentgen examination. Correspondingly there was a more advanced degree of brain development — lobar holoprosencephaly (type A see Table) (ref 7).

Case 6 SD was a 5 1/2 week old male with facial features suggestive of cebocephaly. The occipito frontal circumference was 28 cm. There was no clinical evidence of trigonocephaly. Physical examination revealed the upper lip to be united in the midline but without a distinct philtrum. There was a rudimentary nose and a single aperture to the nasal cavity. There was no cleft palate. The eyes were prominent. Frontal and lateral skull roentgenograms indicated microcephaly. The orbits were shallow and the protruding orbital soft tissues were seen in the lateral projection. The anterior cranial fossa was small. Small nasal bones were identified but the nasal bridge was flat. There was orbital hypotelorism. A midline osseous structure was seen in the frontal projection probably representing the perpendicular ethmoid.

associated with holoprosencephaly. A continuing study is in progress at this institution using laminography of the face and skull to aid in this differentiation.

Chromosome studies suggest that the holoprosencephalies fall into two groups. Both are similar or identical in the craniofacial malformations but differ in respect to the extracephalic anomalies. One group has a minor or perhaps one major extracephalic anomaly. The second group is associated with multiple severe extracephalic anomalies such as malformations of the heart and gastrointestinal tract. The karyotype of Group 1 has been studied in a very limited number of patients and found to consist of the normal number of chromosomes. Group 2 on the other hand may have an additional chromosome of the 13/15 or D group. It appears that the brain malformation can be predicted from the facial configuration regardless of the karyotype (ref. 3, 6). Further chromosome studies will be necessary to confirm this hypothesis.

SUMMARY

Median defects of the face and skull are strongly associated with a brain malformation characterized by incomplete division of the prosencephalon into cerebral hemispheres. A teratologic series can be recognized which when arranged in decreasing order of severity includes five distinct facial types: 1) Cyclopia, 2) Ethmocephaly, 3) Cebocephaly, 4) Median cleft lip and 5) Philtrum premaxilla anlage. Roentgenographic features characteristic of this series include orbital hypotelorism and absence or marked hypoplasia of the median facial structures and crista galli.

ZUSAMMENFASSUNG

Mediane Defekte des Gesichtes und Schädels sind häufig von Gehirnbildungen begleitet. Diese sind dadurch charakterisiert, dass sich das Prosencephalon unvollständig in die Hemisphären teilt. Man kann eine teratologische Serie erkennen, die bei Reihung nach abfallender Schweregrad 5 Typen umfasst: 1) Cyclopie, 2) Ethmocephalie, 3) mediane Lippenspalte, 4) Philtrum, Præmaxillaanlage. Röntgenmerkmale, die für diese Serie charakteristisch sind, inkludieren orbitalen Hypotelorismus und Fehlen oder bedeutende Hypoplasie der medianen Gesichtstrukturen und crista Galli.

RÉSUMÉ

Les malformations médianes de la face et du crâne sont en corrélation étroite avec une malformation cérébrale caractérisée par une division incomplète du prosencéphale en hémisphères cérébraux. On peut établir une série tératologique qui comprend rangés par gravité décroissante cinq types faciaux distincts: 1) Cyclopie, 2) Ethmocephalie, 3) Cébocephalie, 4) Bec-de lièvre médian et 5) ébauche philtrum-prémaxillaire. Les signes radiologiques caractéristiques de cette série comprennent l'hypotelorisme orbitaire et l'absence ou l'hypoplasie marquée des structures médianes de la face et de la crista galli.

and no cases of cyclopia or ethmocephaly. All patients with the median cleft lip facial type and the one case of cebocephaly had the severe brain malformation of lobar holoprosencephaly.

The roentgen findings in median cleft lip facial type include microcephaly, orbital hypotelorism, absence or marked hypoplasia of the median facial bones including the vomer, ethmoid, nasal, premaxilla, and primary palate as well as the nasal septum. The presence of this group of roentgen findings permits the diagnosis of lobar holoprosencephaly and the prognosis of almost certain death within the first year of life (ref. 7). Associated roentgen findings include maxillary bone hypoplasia, shallow orbits with protruding orbital soft tissues, and high orbital roofs with a small anterior cranial fossa and small frontal bones. The longest diameter of the orbits in the frontal projection may be oriented vertically or supero medially and the superior orbital margins may be semi circular ('half moon orbits') in configuration (ref. 1).

Lobar holoprosencephaly is almost always associated with microcephaly, except under the unusual circumstances of a large dorsal sac with a large intracranial fluid volume as seen in Case 3 of this series.

Defective development of the median facial structures and the orientation and configuration of the orbits serve to differentiate patients with microcephaly and lobar holoprosencephaly from other microcephalic patients. In the absence of median facial defects the half moon configuration of the superior orbital contour and the vertical orientation of the longest diameter in the frontal projection help to differentiate patients with microcephaly and hypotelorism from those without hypotelorism. Interorbital distance measurements in infants and children have been obtained based on posteroanterior skull roentgenograms believed to be normal (ref. 2). This scale of measurements relates interorbital distance to age. In the presence of microcephaly, however, these measurements must be applied with great caution.

WELCHER who first named trigonocephaly more than a century ago recognized two types: 1) the simple cranial deformity with no other associated median facial and presumably no brain malformation and 2) the complex form associated with other median facial defects and holoprosencephaly (arhencephaly) (ref. 12). Patients with the mild form of holoprosencephaly may be normal externally or may have trigonocephaly. It is in this latter group that careful evaluation of the midline facial structures may be useful in differentiating simple trigonocephaly from the complex form associated with brain malformations which are not evident clinically. Defective development of the midline components of the ethmoid bone (crista galli and perpendicular plate), vomer, premaxilla, primary palate, nasal bones and nasal septum may serve as a means of differentiating simple trigonocephaly from trigonocephaly

THE CRANIOPHARYNGEAL CANAL

by

R M LOWMAN F ROBINSON and W H McALLISTER

The craniopharyngeal canal arises superiorly in the mid portion of the floor of the hypophyseal fossa and when complete may extend inferiorly to terminate behind the rostrum of the sphenoid between the alae of the vomer. Described by a variety of names i.e. basipharyngeal or basioccipital canal, the nature of this structure has been controversial. One thesis backed by tradition purports the canal to be the pathway of transmission of the buccal hypophyseal diverticulum i.e. the pathway of the former stalk of Rathke's pouch. Another view maintains that this structure represents remnants of a vascular channel formed during osteogenesis by fetal vessels.

During the course of study of hypophyseal embryogenesis and suprasellar tumors a completely patent craniopharyngeal canal was encountered in a disarticulated skull in the department of anatomy (Figs 1 and 2). The following studies were undertaken to investigate the nature of this canal and why it may persist for a short time or permanently.

1 Anatomic studies of the sub human primates and lower mammalian skulls were made.

2 Studies were made of disarticulated sphenoid bones and human skulls in the Department of Anatomy of the Yale University School of Medicine.

3 Sagittal sections of the sphenoid regions of the skulls of stillborn prematures

REFERENCES

- 1 ANDERSON F M, GWINN J L and TODT J C Trigenocephaly. Identity and surgical treatment *J Neurosurg* 19 (1962) 723
- 2 CURRARINO G and SILVERMAN F N Orbital hypotelorism arhinencephaly and trigonocephaly *Radiology* 74 (1960), 206
- 3 DEMYER W A 46 chromosome cetocephaly with remarks on the relation of 13—15 trisomy to holoprosencephaly (arhinencephaly) *Ann Paediat* 203 (1964) 169
- 4 — and WHITE P The electroencephalogram in holoprosencephaly (arhinencephaly) *Arch Neurol* 11 (1964) 507
- 5 — and ZFMAN W Alobar holoprosencephaly (arhinencephaly) with median cleft lip and palate clinical electroencephalographic and nosologic considerations *Confin neurol* 23 (1963) 1
- 6 — — and PALMER C Familial alobar holoprosencephaly (arhinencephaly) with median cleft lip and palate *Neurology* 13 (1963) 913
- 7 — — — The face predicts the brain diagnostic significance of median facial anomalies for holoprosencephaly (arhinencephaly) *Pediatrics* 34 (1964) 256
- 8 LONGMAN J Medical embryology p 303 Williams & Wilkins Baltimore 1963
- 9 MITTLER F A Congenital malformations of the brain *J Neuropath exp Neurol* 6 (1947) 98
- 10 RIEMENSCHNEIDER P A Trigenocephaly *Radiology* 68 (1957) 863
- 11 STARK R B and CHURMAN N A The development of the center of the face with particular reference to surgical correction of bilateral cleft lip *Plast reconstr Surg*, 21 (1958) 177
- 12 WELCKER H Untersuchungen über Wachstum und Bau des menschlichen Schädels p 120 Verlag Von Wilhelm Engelmann Leipzig 1862



Fig 2 Lateral film of a disarticulated sphenoid bone with a thin metal probe inserted to show the course of the canal shown in fig 1. The canal was not visible on conventional lateral films.

basilar surface of the sphenoid approximately at the junction where the wings of the vomer meet the body of the sphenoid bone. The lower opening of the canal in this area may vary in shape. AREY has described a specimen with an elongated funnel shaped foramen in this area which measured 2.0 mm in its greatest diameter. Canals of larger size have also been described. Blind canals may extend into the body of the sphenoid bone from one or both surfaces of this bone ranging from tiny pits to passages sometimes 5.0 mm long.

The contents of the canal have been studied in an attempt to evaluate the character of this structure. The contents have been described as (1) connective tissue continuous with the dura and the periosteum (2) a vein opening into the cavernous sinus above and into a longitudinal periosteal vein below, (3) an artery has been described entering the sphenoid bone and studies made of cat specimens and in addition (4) DRAGER (1944) has described nerve bundles arising from the internal carotid plexus which were distributed to the blood vessels within the sphenoid bone in sections made from cats.

The description of the vascular constituents of the human craniopharyngeal canal is based on studies of injected fetuses made by ARAI (1907). In addition to the structures described above other authors have described accessory hypophyseal tissue in the canals. Hypophyseal tissue within the open canal itself (HABERFELD 1909) as well as pituitary tumors within the body of the sphenoid have been reported (GARSCHIN 1930).

Methods of study and observations

A fund of information concerning the occurrence of the craniopharyngeal canal in the sub-human primates and lower mammals exists. The canal was found in 37% of 321 anthropoid apes studied. In chimpanzees a previous canal occurred in 64% of the specimens (CAVE 1931). Other observers have noted

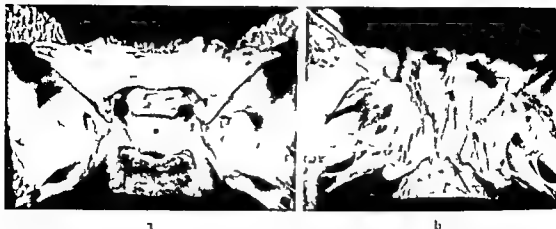


Fig. 1 a) Photograph of upper surface of an adult disarticulated sphenoid bone showing upper end of craniopharyngeal canal in the hypophyseal fossa b) Lower opening of the craniopharyngeal canal

and full term fetuses were examined to determine whether or not residual canals or related structures could be identified

4 Roentgenologic and histologic studies were made of the sphenoid bones of rabbits and cats

5 The incidence of occurrence of the craniopharyngeal canal in approximately 100 skull films or newborn fetuses was evaluated

Various investigators have studied the frequency of occurrence of this canal in varying human age groups. Early descriptions of the canal were recorded by HENLE (1855), VIRCHOW (1857) and LANDZERT (1868). The studies of specimens beyond the age of infancy and the frequency of occurrence of the craniopharyngeal canal have been summarized (AREX 1950, CAVE 1931, SOKOLOV 1904, MAGGI 1890 and CITELLI 1913). AREX noted that the channel occurred at the rate of one specimen in 238 human skulls examined (0.42%). The recorded incidence of complete craniopharyngeal canal is 35 out of 8338 skulls studied. In addition, various observers have pointed out that incomplete canals occurred in this area far more commonly than complete canals. The length of the blind canals extending into the sphenoid bone varies. Closure of a canal tends to proceed progressively from below upwards. Pits occurring on the floor of the hypophyseal fossa were considered the last trace of a former previous canal (Fig. 3).

The proximal aperture of the canal may vary from a circular to an oval outline in the midplane of the deepest part of the sella. The oval foramen measures from 1.0 to 1.5 mm in diameter. The canal, when completely patent, extends downward and backward for a distance of 15 to 16 mm, and emerges on the

formalin solution and then decalcified by overnight immersion in Decal. The blocks were washed in tap water for 24 hours and after immersion in 50% alcohol for 24 hours the blocks were trimmed to a thickness of 5 mm and embedded in Paraplast in the normal manner.

Serial sections were then cut from the embedded tissue and each time 20 sections were cut the six subsequent sections were mounted on glass slides for staining. Thus sections 20 through 26, 40 through 46, 60 through 66 were available for staining. Prior to sectioning roentgenographic studies were made of the midline sagittal sections to determine if patent craniopharyngeal canals could be demonstrated (Fig. 5). Observations were then made on the sections from these blocks of the sphenoid bone to determine the presence of the craniopharyngeal canal. The adjacent structures such as the hypophysis, the hypothalamus, the blood vessels, nerves and pharyngeal tissues were likewise studied. Appropriate stains, i.e. hematoxylin-eosin, Masson and the Weigert's myelin stain to demonstrate the tissues and nerve fibers in this area were made. The study of the sphenoid bone in the group of cat skulls examined, revealed the presence of a well defined canal in every specimen. The canal appeared to be surrounded by a layer of compact bone and extended from the floor of the hypophyseal fossa through the posterior margin of the sphenoid bone to the pharyngeal surface of the skull. In some of the cases the canal did not completely extend to the pharyngeal surface of the sphenoid bone. At the sellar entrance a strand of connective tissue was noted to continue with the sellar capsule and periosteum through the canal toward the inferior surface of the body of the sphenoid. The canal contained a relatively large vein and a rather small artery. Tributaries of these vessels appeared to arise in the sphenoid bone and merged to form the larger vessels (Fig. 7). The majority of the vessels were apparently derived from the internal carotid plexus extending from the circle of Willis into the hypophyseal capsule. No definite demonstration of nerve fibers accompanying the larger arterial branches within the sphenoid bone were shown using the routine stains for myelin.

During the period 1960 to 1964 an unselected group of stillborn fetuses were obtained. Lateral skull films of the fetuses prior to postmortem studies were obtained. In 25 of the fetuses the sphenoid bones were completely removed and sagittal midline sections of the sphenoid bone containing the sella turcica were completed. This portion of the bone was divided in its center and sections were made from both margins of the blocks in an attempt to define the structures in this area. The sections were stained with hematoxylin and eosin. No attempt to dissect the posterior aspects of the sella turcica were made at this time. In addition roentgenographic magnification as well as routine views were made in some cases in an attempt to define the presence of a craniopharynx.

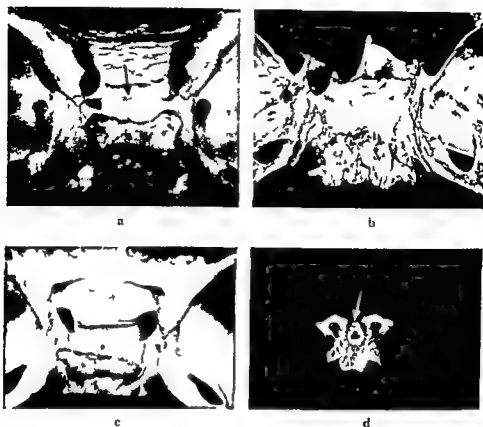


Fig 3 a) Photograph showing upper end of an incomplete craniopharyngeal canal. Small pits in posterior portion of hypophyseal fossa. b) Multiple pits are visible in the floor of the hypophyseal fossa. c) A large ventral pit on the floor of the hypophyseal fossa and smaller pits in the anterior portion of the fossa and region of the tuberculum. d) Photograph of a sphenoid bone of a newborn. A large completely patent craniopharyngeal canal.

the constant recurrence of the canal in cats and rabbits (MAGGI 1893, DRAGER 1944). An examination of a wide range of mammalian skulls in the Peabody Museum collection of the Yale University revealed openings in the floor of the pituitary fossa which in some instances were continuous and could be traced by probe to extend to the under surface of the skull to the vomero sphenoid junction.

Our observations confirm the persistent presence of the canal as a constant feature in the rabbit and the cat in the skull collection of the Museum. Further studies were then undertaken of cat and rabbit skulls. The severed heads of these animals were frozen to minus 10° C and sectioned in the vertical sagittal plane by band saw while still frozen solid. The resulting section containing all of the midline structures was approximately 8 to 10 mm in thickness. The section containing this tissue was fixed for 24 hours in buffered 10 %

formalin solution and then decalcified by overnight immersion in Decal. The blocks were washed in tap water for 24 hours and after immersion in 50 % alcohol for 24 hours the blocks were trimmed to a thickness of 5 mm and embedded in Paraplast in the normal manner.

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general canal. In none of the lateral conventional films was a definite craniopharyngeal canal visible. The studies of the stained sections of the sphenoid bone and the adjacent pituitary structures, however, demonstrated large vascular channels arising superiorly and inferiorly in the body of the sphenoid bone which extended into this structure. While large vascular channels were demonstrated in the region of the sphenoid bone which simulated those described in the cat, a large gross definite channel was not distinguished in any of the cases. However, large vessels arising from the margins of the sellar capsule extending into the region of the sphenoid bone could be demonstrated (Fig. 8).

A review of the collection of skulls in the Department of Anatomy of the Yale University School of Medicine showed one infant skull which had a prominent complete canal (Fig. 3 d). One adult sphenoid bone also demonstrated a complete canal. While the canal was not visible in the routine film the insertion of a wire probe (Fig. 2) demonstrated its course. Several skulls demonstrated incomplete canals which were more common than the completely patent canals (Fig. 3). In other specimens blind pits were demonstrated to extend for several millimeters leading from one or both surfaces of the skull into the body of the sphenoid bone. The number of skulls surveyed was limited and therefore insufficient to provide a valid statistical conclusion. The description of the craniopharyngeal canal and the sellar pits is similar to that which has been noted previously.

The lateral films of 400 newborn skulls were completed and reviewed. Detailed examination of these films revealed a fine line which traversed the body of the sphenoid bone in the non-pneumatized fetal sphenoid in two cases (Fig. 4, a and b). An incomplete line was demonstrated in an additional 2 cases. This thin line was thought to represent the craniopharyngeal canal. The passage of fine vascular markings over the margins of the sella and the sphenoid bone rarely presented problems and were readily differentiated with the aid of a magnifying hand lens. By contrast, the synchondrosis between the presphenoid and the postsphenoid, in addition to the spheno-occipital synchondrosis were frequently seen. The synchondrosis between the presphenoid and postsphenoid or basisphenoid and portions of the body of the sphenoid bone, i.e. the intersphenoidal synchondrosis, occasionally presented the appearance of a canal in the anterior portion of the pituitary fossa. When the articulation between the pre- and postsphenoid segments of the sphenoid bone was demonstrated in properly exposed films it measured 0.5 to 1.0 mm in width and 5.0 mm in length. The anterior superior portion of the synchondrosis terminated just anterior to the margins of the anterior clinoid processes. This synchondrosis was always more sharply defined than the thin line seen in the mid portion of the sphenoid bone. The differentiation of the spheno-

occipital synchondrosis posteriorly posed no problem and was seen in all of the cases studied

The thin line which was demonstrated in two of the cases, and partially in another two cases, was thought to represent the craniopharyngeal canal or a remnant of this structure. It is also possible that this represents the larger vascular channel demonstrated in the histological sagittal sections. Whenever demonstrated it was situated several millimeters posterior to the intersphenoidal synchondrosis. It arose approximately in the midportion of the sellar floor and extended inferiorly for a distance of 3 to 5 mm toward the base of the skull. Although the inferior margins could be outlined by air in the epipharyngeal area, the lower portion of the line in this region was obscured. In one case there appeared to be areas of minimal marginal sclerosis delineating the lateral borders of the canal. The anterolateral temporal fontanel presented no difficulties in differential diagnosis. In addition, however, one must be certain that the overlapping of the linear lines produced by the vascular grooves traversing the anterior portion of the squamous segment of the temporal bone are not mistaken for a persistent craniopharyngeal canal. An anterior branch of the middle temporal artery may produce a similar line in this area on the external skull surface. Meningeal arterial vascular grooves on the endosteal aspect of the skull may occasionally present problems. The coursing of the vessel beyond the margins of the sphenoid bone will aid in differentiation of such vascular grooves.

In addition, routine and special skull film studies in 25 cases of craniopharyngioma were reviewed and examined from the department of neurosurgery. In one of these were structures which could be construed or identified as representing a patent craniopharyngeal canal. In none of the cases examined were craniopharyngiomas found within the margins of the sphenoid bone itself.

Discussion

The demonstration of the craniopharyngeal canal and the sellar pits have prompted this study in an attempt to determine the character of these structures. The rare and infrequent comments on the craniopharyngeal canal in the roentgenographic texts indicate that the craniopharyngeal canal has been passively accepted as the pathway transmitted by Rathke's pouch (CAFFEY 1950 DYKE 1948 KOHLER & ZIMMER 1956 SHAPIRO & JANZEN 1960). The radiographic demonstration of the canal in the base view of the skull was proposed by DYKE in 1948 but anatomic confirmation of this finding was not forthcoming. Our own radiographic attempts which have included the routine positions, laminographic, stereoscopic views in addition to magnification views

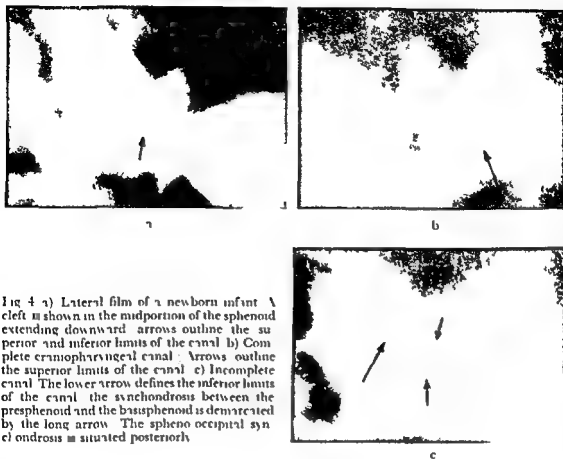


Fig. 4 a) Lateral film of a newborn infant. A cleft \equiv shown in the midportion of the sphenoid extending downward; arrows outline the superior and inferior limits of the canal. b) Complete craniopharyngeal canal. Arrows outline the superior limits of the canal. c) Incomplete canal. The lower arrow defines the inferior limits of the canal; the synchondrosis between the presphenoid and the basisphenoid is demarcated by the long arrow. The sphenoid occipital synchondrosis \equiv situated posteriorly.

have indicated that neither the pits nor the canal are to be defined in the adult skull. Thus far, we have not encountered a patent craniopharyngeal canal in the base view studies. Detailed roentgenographic studies were made on dried skulls following the drilling of 1.0 and 2.0 mm holes through the sellar floor to demonstrate the difficulties in demonstrating the channel. At no time was the canal shown in the lateral views. Moreover, the demonstration of the drilled channels in the base view required special positioning to project the long axis of the canal end on. Though the canal occurs with high incidence in the sphenoids of various mammals, this structure was not shown using the routine radiographic techniques. The histologic studies of the newborn skulls confirmed the presence of vascular channels which could simulate the course of the craniopharyngeal canal. These channels were encountered frequently in the studies of the sagittal sections of the fetal skulls. Studies of these sections at the same time did not reveal any evidences of proliferative nests of squamous or ciliated epithelium or aberrant epithelial invagination.

The development and the constituents of human hypophyseal embryo



Fig 5 a) Lateral film of a section of a cat's skull prepared by sectioning the midportion of the skull after freezing. A 1.5 cm sagittal section containing the midline structures was then cut by a bandsaw. Although a completely patent canal is present, it is not visible in this film. b) Lateral film of sagittal section of a rabbit's skull prepared from a midline segment cut following freezing. This contained all the midline structures. Prominent sphenooccipital synchondrosis posteriorly. (Histologic sections showed a craniopharyngeal channel in the midportion of the sphenoid bone.)

genesis were exhaustively studied by TILNEY (1936). The hypophysis cerebri is derived embryologically from two separate ectodermal structures: the neuroectodermal prolongation from the floor of the primitive forebrain and the ectodermal lining of the primitive stomodeum or the mouth cavity. The latter, known as Rathke's pouch, gives rise to the anterior lobe or the adenohypophysis, whereas the neuroectodermal diverticulum gives origin to the posterior lobe and the infundibulum. It was TILNEY's observation that the craniopharyngeal duct ceased to exist in early embryonic forms and that this structure initially represented a transient communication between the mouth cavity and the pituitary pouch becoming a solid stalk which later disappeared. AREY has pointed out that the elongation and narrowing into a slender stalk and the subsequent disappearance of this stalk begins at 6 weeks (12.0 mm embryo) and is usually obliterated at 8 weeks (23.0 mm embryo) in the future sphenoidal region. Variations of the obliteration of this duct were noted. TILNEY concluded that no substantial proof existed to support the view that the embryonal hypophyseal duct or attenuated stalk persists into adult life in the human. He suggested that there was little to favor the continued use of the term craniopharyngeal duct.

In order critically to assess the sphenoidal channel, it is necessary to correlate the development of the cartilage, bone and blood vessels of the basisphenoidal



Fig. 11 a) Sagittal section through pituitary and midportion of the sphenoid of a rabbit. The section is just to the one side of the midline and does not completely show the posterior lobe of the pituitary gland. A large thin walled venous channel runs in the pituitary capsule down to join a thin walled triangular venous lake in the midportion of the sphenoid bone. b) Midline section from same animal showing the venous lake. The posterior lobe of the pituitary is better shown. 1 — Synchondrosis between pre- and postsphenoid. 2 — spheno-occipital synchondrosis. 3 — venous lake in midportion of sphenoid. A channel connects this to venous channels shown at the inferior margins of the pituitary gland.

1919 The studies by AREY (1950) and MALL (1906) indicate that concurrent with the disappearance of the stalk of Rathke's pouch, there is chondrification of the sphenoid. Precartilaginous changing into cartilage within the body of the sphenoid was shown in the 17 mm embryo. A canal marking the location of the former stalk could no longer be identified in embryos of 33, 37, 55 up to 255 mm. MALL (1906) using cleared specimens demonstrated paired ossification centers in 70 mm embryos and showed that these were united at 105 mm. Thus, in terms of time, the paired sphenoid ossification centers appear at approximately 13 weeks and unite at 16 weeks. Primitive marrow formation with conversion of cartilage into spongy bone was shown in the 110 mm length embryo.

The studies of the sagittal sections of the cat, rabbit and human sphenoid bones presented significant information on the vascular structures of this area. Blood vessels were seen to course lengthwise along the curved depth of the hypophysial fossa adjacent to the perichondrium of the sphenoid bone. In several sections, large prominent vessels were noted beneath the margins of the hypophysis. These arose in the region of the sellar capsule and extended downward into the sphenoid bone. Other sections studied demonstrated a central vascular channel pressing directly downward from the lowest point of the hypophysial fossa and penetrating the body of the sphenoid bone (Figs 6, 7, and 8). The central canal appeared to connect laterally with other smaller vascular channels. In one section at least, vessels beneath the sphenoid bone had extended into the peripheral spongy bone.



Fig. 7



Fig. 8

Fig. 7 Midline section from a rat. The bony canal is evident. Thin walled venous channels are seen in the pituitary capsule and they communicate through this channel inferiorly. Several tiny acini structures lined by cuboidal epithelium and containing a minute amount of colloid like substance which histologically are much like the medulla of the pituitary are also seen. The venous channel in this case is surrounded by cancellous bone.

Fig. 8 Midline section through a full term stillborn human fetus. A large thin walled venous channel runs in the capsule of the pituitary. Such channels communicate with large tortuous venous sinusoids in the midportion of the sphenoid bone.

Thus the channel follows the same topographical course along which the elongating stalk of Rathke's pouch is directed. It is unlikely however that this channel represents the canal through which in early fetal life the hypophyseal diverticulum of the buccal ectoderm is transmitted. The evidence indicates that this residual canal is neither the remnants of the hypophyseal stalk nor the residuum of the space formed by the degenerating hypophyseal stalk. Moreover it does not appear to be the remnants of a persistent channel in the cartilage which has not obliterated during the fusion of the ossification centers. From the observation made from the histologic studies this appears to be a channel formed during osteogenesis when vessels are uniting with the marrow spaces by erupting from the periosteum into the dissolving cartilage and the newly developing bone. BARSON (1942) and AREY (1950) have stressed that the vessels of the craniopharyngeal area and the basisphenoid are situated in portions of the skull that are in line with the vertebrae and that the vertebral bodies are pierced dorsoventrally by vessels that simulate similar vessels. Reference to the basi vertebral veins has been made by previous anatomists and reemphasized by BARSON (1942) and WAGNER & PENDEGRASS (1939). The vessels which occur in the craniopharyngeal area are contained in the paleocranium that is situated in line with the vertebrae. The vessels in these areas may therefore not only be comparable they may even be homologues.

Speculation as to why the canal appears and persists apparently depends on the factors which control the appearance, the location and the persistence of nutrient foramina and channels with bones.

Conclusion

The craniopharyngeal canal is an anatomic finding in the mammalian skull, found with decreasing incidence as one ascends the phylogenetic scale. In the majority of mammals the craniopharyngeal canal closes soon after birth. The passage however may persist throughout life in some species. Although patent and complete canals are rarely found, our studies indicate that the canal can usually not be demonstrated utilizing routine and special radiographic techniques. Even when patent canals are present in the disarticulated skull, this structure was not adequately demonstrated. The inability to show this conduit is probably due to the small size of the canal, the location of the sphenoid in the adult bone, and the difficulties in positioning the skull for radiographic demonstration of this structure in the base view.

A thin obliquely vertical line of reduced density traversing the midportion of the sphenoid was encountered in the lateral view studies of newborn skulls. Corresponding to the channels shown in the sagittal histologic sections, this was considered to represent the craniopharyngeal canal. This line or segments of such a line were roentgenologically demonstrated in 4 cases out of 400 skull films studied. The unmineralized character of the newborn sphenoid probably accounts for the roentgenographic demonstration of the canal or segments of this structure. In 2 of these cases the vertical line did not completely traverse the body of the sphenoid.

In various species of animals despite the known presence of the canal, our studies indicated that the structure could not be shown in the lateral projection in specially prepared specimens.

The canal is not to be confused with the normal neonatal synchondroses occurring in the sphenoid bone. Histologic studies of the sphenoid bone of various premature and term stillborn fetuses showed no squamous cell arrest or cystic glandular structures suggestive of hypophyseal duct origin in the sphenoid bone. The histologic studies made of sagittal sections of the sphenoid bone in cats and rabbits demonstrated persistent craniopharyngeal canals. Vascular structures in the canal were demonstrated in these sections. One animal demonstrated connective tissue lined by cuboidal epithelium similar to that of the median lobe of the pituitary. Myelinated nerve fibers were not seen in either animal species. Although various theories exist concerning the anatomic and embryologic significance of the craniopharyngeal canal, our

studies indicate that this probably represents the residuum of the canal formed during osteogenesis when vessels are connecting and are being distributed through the marrow spaces by erupting from the periosteum into the fetal cartilage and the adjacent developing spongy bone. The mechanism of the development of this canal is much the same as the formation of the channels occupied by the basivertebral vein in the body of a vertebra. This conduit for vessels and nerves has been previously interpreted in the past as the pathway of the former stalk of Rathke's pouch. While the hypophyseal stalk is obliterated early, the later appearing craniopharyngeal canal pursues the same general direction. Since the present designations describing this structure imply a neurogenic embryonic origin derivation these should be discarded. More appropriate, would seem to be the use of the term intrasphenoidal channel.

Acknowledgement

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SUMMARY

The craniopharyngeal canal is an anatomic finding in the mammalian skull found with decreasing incidence as one ascends the phylogenetic scale. The channel is rarely encountered in human infancy. This channel for vessels and nerves previously considered the conduit for the former stalk of Rathke's pouch is rarely demonstrated roentgenographically. The canal when it persists is apparently formed during embryologic osteogenesis and vascularization of the body of the sphenoid bone.

ZUSAMMENFASSUNG

Der Craniopharyngealkanal wird normalerweise am Mammalia-Schädel gefunden. Er kommt jedoch in abnehmender Frequenz vor je höher die phylogenetische Entwicklung ist. Beim menschlichen Kinde findet man ihn selten. Der Kanal für Gefäße und Nerven und ursprünglich für den Stiel der Rathkeschen Tasche vorgesehen ist röntgenologisch selten darstellbar. Beim Fortbestehen des Kanals wird er offensichtlich während der embryonalen Osteogenese und Vaskularisierung des Keilbeinkörpers gebildet.

RÉSUMÉ

On trouve d'autant moins fréquemment le canal craniopharyngien dans le crâne des mammifères qu'on s'élève dans l'échelle phylogénique. Ce canal existe rarement chez le nourrisson humain. Ce canal destiné à des vaisseaux et à des nerfs et considéré autrefois comme destiné à la tige de la poche de Rathke est rarement mis en évidence radiographiquement. Quand il persiste, il paraît formé au cours de l'ostéogenèse et de la vascularisation embryologique du corps de l'os sphénoïde.

REFERENCES

- ARAI H. Der Inhalt des Canalis craniopharyngeus. *Anat. Hefte* 33 (1907) 411
- AREY L. B. The craniopharyngeal canal reviewed and reinterpreted. *Anat. Rec.* 106 (1950) 1
- BATSON O. V. Vertebral veins in metastatic processes. *Ann. intern. Med.* 16 (1942) 38
- Vertebral vein system as a mechanism for the spread of metastases. *Amer. J. Roentgenol.* 48 (1942) 715
- CAFFRY J. *Pediatric x-ray diagnosis*. Year Book Publishers, Chicago 1950
- CWEL A. J. F. The craniopharyngeal canal in man and mammal. *J. Anat.* 64 (1941) 363
- CITELLI I. Sur la fréquence relative du canal craniopharyngien chez les enfants et les jeunes gens et sur l'importance de ce fait pour ma théorie. *Ann. Mal. Oreil. Larynx* 39 (1913) 338
- DRACER C. A. Nerve fibers within the craniopharyngeal canal. *Anat. Rec.* 88 (1944) 235
- DYER C. G. Persistent craniopharyngeal canal in diagnostic roentgenology. Ed. by R. Golden, p. 30f. Williams & Wilkins Co. Baltimore 1946
- GARSCHIN W. Ein Fall der intrasphenoidalen Hypophysengangsgeschwulst. *Z. Krebsforsch.* 31 (1930) 432
- HANDREFFID W. Die Rachenröhrenhypophyse, andere Hypophysengangsgeste und deren Bedeutung für die Pathologie. *Beitr. path. Anat.* 46 (1909) 133
- HENLE J. *Handbuch der systematischen Anatomie des Menschen*. Vieweg und Sohn, Braunschweig 1. part 1 (1855) 283
- KOHLER A. and ZIMMER F. A. *Borderlands of the normal and early pathologic in skeletal roentgenology*. Grune & Stratton, Inc. New York 1956
- LANDZERT T. Ueber den Canalis craniopharyngeus am Schadel des Neugeborenen. *St. Petersb. med. Ztschr.* 14 (1868) 133
- MAGGI L. Intorno al canale craniofaringeo in alcuni roicanti. *Rivista Ist. Lombardo di scienze e lettere (Rendiconti)* Ser. 2 23 (1890) 729
- MALL F. P. On ossification centers in human embryos less than 100 days old. *Amer. J. Anat.* 5 (1896) 433
- RATHKE H. Nachträgliche Bemerkungen zu dem Aufsätze über die Entstehung der Glandula pituitaria. *Arch. f. Anat. Physiol. und wissenschaftliche Medizin von Müller* 1839
- SITAPIRO R. and JANZEN A. H. *The normal skull: a roentgen study*. Paul B. Hoeber, Inc. New York 1960
- SOKOLOV P. Der Canalis craniopharyngeus. *Arch. f. Anat. u. Physiol. Anat. Abh.* (1904) 71
- TILNEY F. *Bull. neurol. Inst. N. Y.* 5 (1936) 387
- VIRCHOW R. *Untersuchungen über die Entwicklung des Schädels, Kundes etc.* Reimer, Berlin 1857
- WAGONER G. and PFENDERGRASS E. P. The anterior and posterior notch shadows seen in lateral roentgenograms of the vertebra in infants. *Amer. J. Roentgenol.* 42 (1939) 663

OBSERVATIONS ON CRANIOLACUNIA

by

DONALD L. McRAE

Craniolacunia also called lacunar skull or Luckenschädel is sometimes considered a cranial dysplasia of no practical importance. However, long term follow up of patients born with this condition shows it to be of importance in prognosis. Patients with craniolacunia have high morbidity and mortality due to associated lesions of the central nervous system and to the secondary effects of the neurological lesions (VOGT & WYATT 1941). Craniolacunia is frequently associated with myelomeningocele and encephalocele less often with simple meningocele. Patients with craniolacunia frequently develop hydrocephalus due to stenosis of the aqueduct of Sylvius and/or to the Arnold Chiari malformation. They are frequently found to have paraplegia deformities of the legs and feet, and urinary and fecal incontinence.

Craniolacunia is characterized by groups of round, oval or finger shaped pits in the inner surface of the cranial vault, separated by slender ridges of bone (Fig. 1a).

The deepest pits may penetrate the full thickness of the vault and become palpable. The pits and ridges are most obvious in the thickest parts of the frontal, parietal and occipital portions of the vault. The newer and thinner portions of these bones are less often and less markedly involved. In the occipital squama the lesions tend to be concentrated just above the internal occipital protuberance with their long axes in a parasagittal direction and the deepest



Fig 1 Male aged 20 days born with a lumbosacral myelomeningocele. Lacunae + + + are seen in (a)



Fig 2 Female aged 2 1/4 months admitted because of an enlarging head no meningocele of any type could be found. Ventriculography disclosed stenosis of the aqueduct of Sylvius and a ventriculoatrial shunt (Torkildsen procedure) was carried out. The head continued to enlarge but at a slower rate. She is reported to have died at home in her second year cause unknown. Lacunae + +

part of the pit lying against the attachment of the tentorium (Fig 1b). The lacunae are present for a few months in fetal and postnatal life, run a predetermined course and then disappear, never to reappear.

Skull and spine films of all patients indexed as cranioleak, encephalocele, myelomeningocele and meningocele were reviewed. Sixty nine cases of cranioleak were found. Fifty eight infants with encephalocele, myelomeningocele or meningocele had undoubted cranioleak. Ten others showed probable



Fig 3 Female aged 1 month born with a small head and a large occipital encephalocele. The sac and its contents were excised but the baby died a few hours after operation. Lacunae +++ Gas has been injected into the sac to see if communication between the sac and the cranium could be established.

craniolacunia. The latter were all less than 3 months of age and had lacunae or impressions on the thickest parts of the frontal parietal and occipital bones with intervening normal bone. One infant 2 1/2 months old with hydrocephalus but without an encephalocele or meningocele had undoubted craniolacunia (Fig 2).

There were 17 patients with encephaloceles 2 with cranial meningoceles 8 with spinal meningoceles 41 with spinal myelomeningoceles and 1 who had hydrocephalus without meningocele. There were 37 females and 32 males.

The lacunar markings on the skull were scored on a scale of four ++++ = lacunae deep sharp edged and numerous involving all parts of the vault +++ = lacunae deep sharp-edged, but involving only the thickest parts of the frontal parietal and occipital bones ++ = definite lacunae but with bevelled edges + = lacunae more or less resembling convolutional markings but present only in the thick parts of the frontal parietal and occipital bones with intervening normal bone and present within the first 3 months of life.

For purposes of further analysis the 4+ and 3+ cases were grouped as marked craniolacunia the 2+ cases as moderate and 1+ as slight.



Fig 4 Male aged 4 days. Lacunae + + + +. The occipital encephalocele was excised. No signs of increased intracranial pressure.

The essential cause of the lacunae is not exactly known. The lacunae are on the inner surface of the vault and do not seem to occur in the base. Since the bones of the base are of endochondral origin, it seems that cranio-lacunae is a dysplasia of bone of intramembranous origin. The frequent occurrence of cranio-lacunae in the upper half of the occipital squama (the intramembranous part) and their almost complete absence in the lower half of the occipital squama (the endochondral part) supports this hypothesis. At post mortem, one finds thickened dural bands over the bony ridges. The dura is thinner in the lacunae than elsewhere, but it is not clear whether this is a primary or a secondary thinning of the dura.

There are several reasons for doubting that the lacunae represent convolutional impressions. Cranio-lacunae is found in infants with low intracranial pressure (Fig. 3).

Five of the 69 children in this series had low intracranial pressure as shown by an abnormally small skull with open sutures. In all five this was due to the presence of a large encephalocele. Four of these children had marked cranio-lacunae and one had lacunae of moderate degree. Cranio-lacunae is found in infants who have no sign of increased intracranial pressure before or after birth (Fig. 4). Cranio-lacunae is found in fetal life and early infancy before brain markings are expected. Occasionally in normal new born infants a few faint brain markings are seen on the top of the vault, probably due to the head down position in utero. However, brain markings usually do not appear in normal children until 1 or 2 years of age and then are most obvious on the



Fig 5 Female aged 10 day born with a lumbosacral myelomeningocele and paraplegia Lacunae
++

lower and posterior parts of the cranial vault as well as on the base. These are not the common areas for lacunar markings. In hydrocephalic infants brain markings are rare before 3 months of age and when they occur they are usually generalized. In children with premature synostosis of the cranial sutures brain markings are rare before the age of 2 months and when present are most marked where the brain is pushing hardest on the bone.

Diagnostic criteria of the condition The age at which the lacunae appear and disappear is not known accurately for several reasons. The lacunae begin to appear when the infant is in utero where the skull is not readily examined. I suspect that in some infants it appears soon after the cranial vault begins to ossify, i.e. soon after the eighth week of fetal life. I have seen marked cranio-lacunia in infants in utero at an estimated age of 7 1/2 months. It has been reported as early as 8 months (HARTLEY & BURNETT 1943, MAIER 1934). Since the depth and number of the lacunae vary from case to case the ease with which it can be recognized in roentgenograms of the mother's abdomen varies. Radiography of the pregnant female is not often carried out in the second trimester of pregnancy and therefore we have no large series of cases for accurate estimation of the age at the appearance of the lesions or the resolution.

The age at which the lesions disappear varies. Some children are born with lesions already fading (Fig 5). Five slight cases were less than 1 month old. It can be assumed that their lacunae were most marked in utero. The other six



Fig 6 Female born with a small sub occipital meningocele and a Klippel Feil deformity. Films at ages 1 week and 1 month show fading of the lacunae to resemble brain markings. No sign of increased intracranial pressure. At age 9 years her skull was normal in size and except for the Klippel Feil deformity she was considered normal.



Fig 7 Female born with a lumbar myelomeningocele and paraplegia. The head enlarged rapidly but was controlled by a shunt. At age 3 1/2 years she was paraplegic, incontinent and showed some mental retardation. Films at 3 days and 2 months.

slight cases were over 1 month of age. Their lacunae were probably most marked about the time of birth.

The lacunae seem to run a pre determined course, a course not altered by low, normal or high intracranial pressure. In some cases the sharp edges of the lacunae became bevelled in a month or two so that they resembled brain markings (Fig 6). Occasionally the lacunae became unrecognizable in this length of time (Fig 7). In other cases there was little change in two or three



Fig 8 Female born with a lumbosacral 1 myelomeningocele very little movement of the legs saddle anesthesia and relaxed anal sphincter Skull films at 14 days and 4 1/2 weeks of age showing little change in spite of increased intracranial pressure

months (Fig 8) There were no definite lacunae after 6 months of age Some reported cases of cranio-lacunia in adolescents and young adults are actually examples of prominent brain markings

Any infant skull with impressions on the inner table of the vault before the age of 4 months should be considered as a possible case of cranio-lacunia, especially if the markings are high and anterior on the vault

Incidence of congenital anomalies The commonest anomalies associated with cranio-lacunia are myelomeningocele encephalocele and simple meningocele Only ten of the patients with cranio-lacunia had simple meningoceles while 58 had either encephaloceles or myelomeningoceles VOGT & WYATT (1941) gave the incidence of cranio-lacunia in patients with all types of meningoceles as 43 % They found cranio-lacunia in 12 % of simple meningoceles and in 53 % of myelomeningoceles

Twenty eight of the 49 cases in this series who had cranio-lacunia and spinal myelomeningocele or meningocele were paraplegic Some of the remaining 21 had a monoplegia and others in whom leg movements were present had sphincter paralysis with saddle anesthesia Fourteen had unilateral or bilateral club foot Anomalies of the ribs were common when the spinal lesion was in the thoracic region Eight patients in the total series showed the Klippel Feil deformity and all eight had encephaloceles myelomeningoceles or meningoceles Diastematomyelia was present in at least four and probably in more Miscellaneous abnormalities were dextrocardia inguinal hernia dislocated hips and squint



Fig 6 Female born with a small sub occipital meningocele and a Klippel Feil deformity. Films at ages 1 week and 1 month show fading of the lucunae to resemble brain marking. No sign of increased intracranial pressure. At age 11/2 years her skull was normal in size and except for the Klippel Feil deformity she was considered normal.



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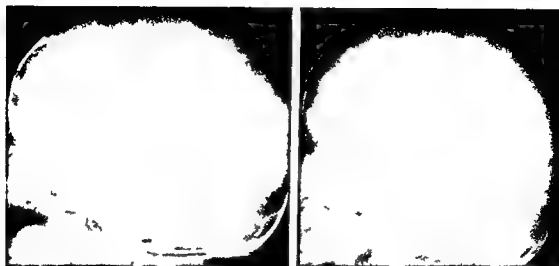


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Thirty two patients had a clearly visible synchondrosis between the pre sphenoid and post sphenoid portions of the sphenoid bone. This is much more frequent than in normal new born children and may be evidence of immaturity of the part of the skull derived from cartilage.

Prognosis. Some of these patients came from distant provinces or other countries and some were treated here many years ago. It was difficult to trace all the patients. The approximate morbidity and mortality can be seen in the Table.

Twenty three patients were alive two or more years after birth. Nine were of normal intelligence and physically were fairly normal. Included in this group were children with Klippel Feil deformity and also club feet. Seven showed some degree of mental retardation usually with some other physical abnormality. Seven were paraplegic but two of these were walking with bilateral leg braces and crutches. Since the last visit of some of these patients was many years ago some may have died since from urinary tract infections or from hydrocephalus.

Five are presumed to be alive since they were discharged from the hospital in infancy with no sign of hydrocephalus or paraplegia and have not returned.

Ten patients are known to have died, mostly in the first year of life. One was the hydrocephalic patient without meningocele who died at the age of two. One was a retarded paraplegic who had a cord bladder and died of uremia at 8 years of age.

Sixteen are presumed to be dead as they were discharged from the hospital in infancy without treatment. In most cases this was because of a combination of rapidly developing hydrocephalus and a severe spinal lesion with paraplegia.

We were unable to trace 15 patients whose encephalocele, meningocele or myelomeningocele was repaired satisfactorily and who had either paraplegia alone or hydrocephalus alone which had been treated by shunt operation. They are listed as questionable.

Conclusions

Congenital anomalies are frequent in these patients.

The lacunar markings in the skull appear in fetal life in the membranous part of the cranial vault. They are grouped in the thickest parts of the frontal parietal and upper occipital squamæ. The lacunæ begin to fade around the time of birth and are usually unrecognizable by the age of 4 to 6 months. They are not related to increased intracranial pressure, and were in fact marked in patients who had low intracranial pressure.

Table

Prognosis in craniolecinia

Incidence	Age at death	Presumed dead	Presumed alive	Presumed alive	Age last visit
<i>Encephalocele</i>					
<i>17 cases</i>					
Marked	1 month	Two	Two	One	0
7 cases	14 days				
Moderate	2 months	One	Two	Two	17 yrs Normal except squint
11 cases					14 yrs Klippel Feil I Q 56
Slight	0	0	0	0	7 1/2 yrs Normal except Klippel Feil
2 cases					11 yrs Normal
<i>Meningocele</i>					
<i>2 cranial</i>					
<i>8 spinal</i>					
Marked	0	Two	Two	0	5 yrs Klippel Feil I Q 90
5 cases					
Moderate	0	0	0	0	11 yrs Normal except Klippel Feil
2 cases					12 yrs Seizures moderate retardation
Slight	0	0	0	0	16 yrs Normal
3 cases					11 yrs Normal except club foot
					7 yrs Normal
<i>Myelomeningocele</i>					
<i>41 cases</i>					
Marked	4 months	Nine	Four	One	3 1/2 yrs Large head normal intelligence
21 cases	13 days				4 1/4 yrs Paraplegia slight retardation
	2 months				3 yrs Paraplegia seizures
					4 yrs Paraplegia large head
Moderate	3 months	Two	Two	Two	5 yrs Large head normal intelligence
13 cases	1 year				12 yrs Club foot I Q 72
					2 yrs Paraplegia large head
					16 yrs Paraplegia I Q 63
					3 1/2 yrs Paraplegia pyelonephritis
					2 yrs Club foot large head
Slight	8 years	0	Three	One	17 yrs Normal except club foot
7 cases					7 yrs Paraplegia slightly retarded
					9 yrs Normal except club foot
<i>No meningocele</i>					
<i>1 case</i>					
Moderate	2 years	0	0	0	0
Totals	10	16	15	5	23

RADIOLOGICAL LOCALIZATION OF THE PINEAL GLAND

by

MARCELO H. MOREAU

There are a number of methods for the determination of displacement of the pineal calcification. The method most generally used is the Vastine Kinney or the Dyke modification which is of great value but presents certain difficulties in the way in which the occipital points are marked particularly the inferior one.

To obviate this disadvantage we propose another method of localization based on a review of 250 cases. Using the lateral film of the skull we propose to relate the calcified pineal body to the *nasion sella turcica (middle center) plane*. We recommend that the film be perforated with a pin at the nasion in order to set a fixed point for measurement of this plane.

Then the occipital angle is determined. This is formed by the nasion sella turcica plane with another plane extending from the nasion to the inferior occipital point (see figure). The values of this angle are A 10 B-14 C 18 , D 22 E 26.

Subsequently the *pineal angle* is obtained above the nasion sella turcica plane which arises from the center of the sella turcica. Values are A-42 , B 38 C 34 D 30 E 26 (see figure).

A transparent film localizer is used which when placed upon the radio

Ten are known to have died and 16 are presumed to be dead, a mortality of 37 % of all patients and a mortality rate of approximately 50 % of all those in whom a reasonable judgment of the outcome could be made. Twenty-three are known to be alive and 5 are presumed to be alive, a survival rate of approximately 10 % of all cases or approximately 50 % of all cases in whom a reasonable decision as to the outcome was possible. Of the 23 known to be alive, 11 are normal or fairly normal.

All children born with spinal anomalies should have skull films made. If craniolacunaria is shown, the chance of the child having severe and/or multiple lesions of the central nervous system is great.

SUMMARY

Sixty-nine patients with craniolacunaria are reported. Multiple often severe lesions of the central nervous system are frequent in this series.

ZUSAMMENFASSUNG

Es wird über 69 Patienten mit Craniolacunien berichtet. In dieser Serie sind häufig, multiple oft ernste Schäden des ZNS vorhanden.

RÉSUMÉ

Présentation de 69 cas de crâne lacunaire. Les lésions multiples souvent graves du système nerveux central sont fréquentes dans cette série.

REFERENCES

- HARTLEY J. B. and BURNETT C. W. F. The radiological diagnosis of craniolacunaria. *Brit. J. Radiol.* 16 (1943) 99.
 MAIER R. J. Prenatal diagnosis of lacuna skull. *Radiology* 23 (1934) 615.
 VOGT F. C. and WILATT G. M. Craniolacunaria (Luckenschädel). *Radiology* 36 (1941) 147.

We believe that this method of localization has an accuracy which is probably similar to that of the Vastine Kinney Dyke method

From our experience it appears that the greater the pineal angle the smaller is the occipital angle and vice versa

SUMMARY

The author proposes to determine the position of the calcified pineal gland in lateral roentgenograms of the skull by its relationship with the nasion sella turcica (middle center) plane. The occipital angle is determined as well as the corresponding pineal angle. This indicates roughly the position of the pineal gland. The sella skull diameter divided by 2.6 indicates the position of the gland more accurately.

ZUSAMMENFASSUNG

Der Verfasser schlägt vor auf Seitenaufnahmen des Schädels die Lage der verkalkten Zirbeldrüse unter Bezugnahme auf die Nasion Sella Linie zu bestimmen. Der Occipitalwinkel und der entsprechende Pinealwinkel werden gemessen. Diese bestimmen annähernd die Lage des Pinealkörpers, die dann mit grosserer Genauigkeit festgelegt wird indem man den Abstand von der Sella zum Schädel durch 2.6 teilt.

RÉSUMÉ

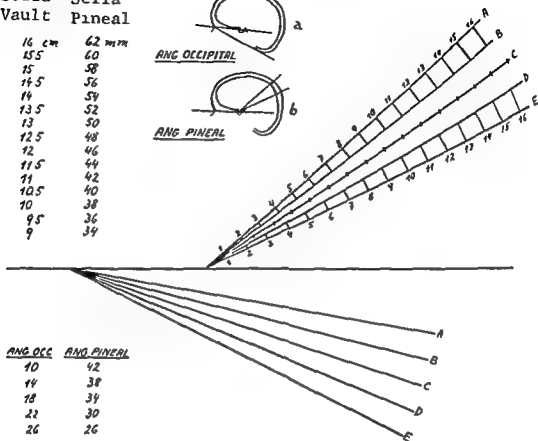
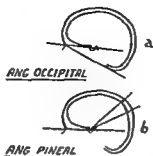
L'auteur s'est efforcé de déterminer la position de l'épiphyse sur les radiographies de profil du crâne par rapport au plan nasion selle turcique. Il a mesuré l'angle occipital ainsi que l'angle pinéal correspondant sur lequel ou près duquel doit se trouver la glande pinéale. Le diamètre selle turcique toute divisé par 2.6 indique la position de la glande.

REFERENCES

- DYKE C. C. Indirect signs of brain tumors as noted in routine roentgen examinations of placement of pineal shadow. *Amer J Roentgenol* 23 (1930) 598
- EPSTEIN B. and DAVIDOFF L. An atlas of skull roentgenograms. Lea and Febiger Philadelphia 1953
- FRAY N. W. A roentgenological study of pineal orientation. A comparison of methods used in pineal orientation. *Amer J Roentgenol* 39 (1938) 899
- GEFFEN A. A new ruler graph for localization of the pineal body. *Amer J Roentgenol* 73 (1955) 118
- PANCOAST H. PENDERGRASS E. and SCHAEFFER P. The head and neck in roentgen diagnosis. Charles C. Thomas Springfield Ill 1940
- VASTINE J. H. and KINNEY K. K. Pineal shadow as aid in localization of brain tumours. *Amer J Roentgenol* 17 (1927) 320

Sella Sella
Vault Pineal

16 cm	62 mm
15.5	60
15	58
14.5	56
14	54
13.5	52
13	50
12.5	48
12	46
11.5	44
11	42
10.5	40
10	38
9.5	36
9	34



	<u>ANG OCC</u>	<u>ANG PINEAL</u>
A	10	42
B	14	38
C	18	34
D	22	30
E	26	26

The figure is drawn on a transparent film. When superposed upon the radiographic film, as indicated in *a* and *b*, the occipital angle, pineal angle, and the ideal position of the pineal gland may be determined.

graphic film as indicated in *a*, allows measurement of the occipital angle (A, B, C, D or E). Then the localizer is moved as shown in *b* of the figure and observe the pineal angle (A, B, C, D, or E), in the same letter of the occipital angle which has previously been measured. The lines representing the various angles of the pineal angle are marked in centimeters (see figure).

The pineal usually falls over one or near one of the lines of the various angles or it may be as much as 7 mm forward and up or 7 mm back and down.

The pineal distance to the center of the sella turcica is obtained by dividing (on the pineal angle line) the distance from the center of the sella to the middle of the vault by 2.6 (see *b* in the figure). The distance of the pineal is indicated in the upper left corner of the pineal localizer; the varying sella turcica-pineal distance in relation to the variation in sella turcica vault diameter can be readily appreciated. These distances are plus or minus 5 mm.

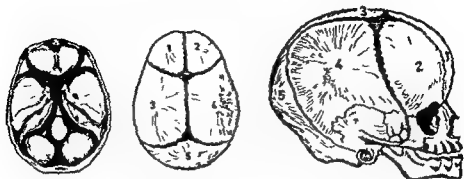


Fig. 1 Basic buttresses and situation of the ossification centers of the vault of the skull

Fig. 2 Situation of the sutures following the main direction of the basic buttresses

2 between the falx and the continuation of the sphenoid wings and pyramids both the *parietal* points,

3 between the pyramids behind the *single occipital* point

The ossification of the vault proceeds with the continued growth of the brain. The bone beams extend along the vascular pattern until the borders of bone centers come into contact with each other. There the sutures are formed. The sutures are consequently situated in the continuation of the above named buttresses (Fig. 2).

We are now able to make the correlation of these observations in normal development by radiological investigations of skulls with a disturbed development—the so called oxycephalics.

Exact data for the vault and base deformity have been gained from various lines and angles obtained from 20 roentgenograms of patients afflicted with such an anomaly. Data of 200 normal skulls have been compared with these. The characteristic deformity of the vault known since the days of HIPPOCRATES (in German referred to as *Turmschädel*) must be combined after the findings of VIRCHOW with a severe deformity of the base.

In the axial projection which offers the best view of the base, a typical deviation from the normal proportions is evident. The sphenoid wings form a greater angle than normal. The axes remain in a more transverse direction. The exact measurements of these angles are: sphenoid angle, normal 90°; sphenoid angle in oxycephaly 120°. The statistical significance is very high with 17 of 16 (Figs 3 and 4).

With increase of the sphenoid angle the axis of the eyes also changes. The

MAIN PRINCIPLES IN DISTURBED DEVELOPMENT OF THE SKULL

Radiological observations

by

HERMANN SCHMIDT

Connections between the base and the vault of the skull in disturbed development have been known since the time of VIRCHOW (1857). This author stated that there is 'no deformity of the vault without deformity of the base'. Later investigations independent of VIRCHOW'S observations, and made by anatomists, have made these connections more evident.

During embryonal development the ossification of the skull begins early in the third month. The first centers appear in the base. The vault is stretched over the brain. When the pressure of the growing brain has reached a certain degree the ossification of the vault begins. The first ossification centers appear at the points of highest growth pressure.

The base as the foundation of the brain also forms the transverse buttresses for the vault of the skull. The main transverse buttresses are the pyramids and the sphenoid wings, the sagittal buttress is the falx cerebri. These buttresses bear the growth pressure of the brain, between them, however, must be the highest scale of pressure. Therefore the first five centers of ossification arise (Fig. 1)

1. between the falx and the continuation of the sphenoid wings both the *frontal* points,



Fig 5 Oxycephaly Short frontal and parietal bone



Fig 6 Situation of the ossification centers in oxycephaly (after KIEPING)



Fig 3 Sphenoid angle in a normal adult



Fig 4 Sphenoid angle in oxycephaly

view the eyes are turned aside. GRAC says in his description of hypertelorism: 'He looks like a hare. As a result we have a divergent strabismus.'

Understandably, the hypertelorism — that is the increased distance between the eyes — depends on this lateral movement of the sphenoid wings. The ethmoid cells become enlarged.

The cranial changes in oxycephaly are well known. Apart from the tower-like deformity there is lack of bulging and shortening of the frontal bone, and as a rule the coronal suture is closed (Fig 5). Measurements prove that in oxycephaly there is regularly not only a short frontal, but also a short parietal bone. The sphenoid angle is normally 95°, in oxycephaly 115°, the angle Endobregma-Tuberculum sellae-Indolimbda is normally 69°—17.6 FE—, in oxycephaly 56°—15.8 FE (FE = Flächeneinheit).

The lateral displacement of the sphenoid wings, as the buttresses for the vault in the skull development, is combined with the shortening of the frontal and parietal bone and the closed coronal suture.

The architecture of bones and the localization of the bone centers may be observed in childhood. In oxycephaly the frontal and parietal centers are situated extremely close to each other, so close that both centers may have

ARTERIAL SPASM IN SUBARACHNOID HAEMORRHAGE

A clinical and experimental study

by

J M ALLCOCK

The records of all patients admitted to Victoria Hospital since 1952 with a diagnosis of subarachnoid haemorrhage have recently been studied and the facts and conclusions obtained from this survey have been given in two papers (ALLCOCK & DRAKE 1963 and in publication)

The first of these showed the necessity of postoperative angiography as a routine procedure. Without angiography one knows little as to the cause of variations in the clinical state and it is impossible to assess honestly the effectiveness of different surgical techniques. It was found for instance that in some patients in whom the surgeon had felt confident at the time of operation that the sac had been completely obliterated there was in fact a significant portion of the aneurysm still filling with contrast. In others a major vessel had been occluded. Many of these patients were doing well and without the post operative angiography would have been recorded as surgical successes.

However the major conclusion was that arterial spasm was one of the main causes of post operative morbidity and mortality. The second study was undertaken to find any factors that might be causing this spasm.

There were 195 patients in whom the source of a subarachnoid haemorrhage

been united from their inception. Only GREIG has observed this peculiarity in adults and has described it as the pterio sphenoic ankylosis in oxycephaly.

The connection with the base deformity is now understandable. If the sphenoid wings stay too far apart from each other, one must expect an approach of the frontal and parietal centers as a result of the forementioned dependence of situation between the buttresses of the base and the ossification centers. This is the reason for the premature closure of the coronal suture. It is a consequence of, not the reason for, oxycephaly.

We have named the characteristic base deformity 'frontal dysplasia', because it concerns the frontal groove and above all the frontal bone. There are still other signs of frontal dysplasia which will not be mentioned here.

Frontal dysplasia and oxycephaly are dependent on each other. With this interpretation the disturbed development fits the anatomic observations of the skull, and also VIRCHOW's statement 'no deformity of vault without deformity of base'.

SUMMARY

Measurements from roentgenograms in 20 cases of oxycephaly compared with the same measurements in 100 normal cases are presented. The typical appearances of the vault in oxycephaly depend on the primary deformity of the base of the skull.

ZUSAMMENFASSUNG

Röntgenbild Schädelmessungen bei 20 Fällen von Oxycephalie werden mit denen von 100 Normalfällen verglichen. Das typische Aussehen der Kalotte bei Oxycephalie hängt von der primären Deformität der Schädelbasis ab.

RÉSUMÉ

Présentation de mesures faites sur des radiographies dans 20 cas d'oxycéphalie comparées aux mêmes mesures dans 100 cas normaux. L'aspect typique de la voûte du crâne dans l'oxycéphalie dépend de la déformation primitive de la base du crâne.

REFERENCES

- GREIG D. M. Oxycephaly. *Fdnb. med. J.* 33 (1926) 189, 280, 357.
 RIEPING A. Zur Pathogenese des Turmschädels. *Dtsch. Z. Chir.* 148 (1910) 1.
 VIRCHOW R. Untersuchungen über die Entwicklung des Schädelgrundes. Reimer, Berlin 1857.



Fig 3 a) Control film b) Narrowing of left internal and external carotid arteries and branches 1 min after injection of serotonin on left c) Normal appearances 8 min later

was shown by angiography to be a ruptured aneurysm, of whom 143 underwent surgery. Post operative angiography was carried out in 92 of these and 38 or 41% demonstrated evidence of arterial spasm.

One factor that seemed to predispose to this was early operation. In those showing post operative spasm the average period between the last haemorrhage and surgery was 5.4 days compared with 11.1 days in those who did not.

Both hypothermia and hyperventilation during surgery had a bad influence. Without cooling 27% showed spasm later while in the group on whom hypothermia was used this figure rose to 50%. With normal ventilation the incidence of spasm was 35% and in the patients who were hyperventilated the proportion showing spasm climbed to 67%. If both were used, spasm was seen later in 86%.

Artificial hypotension during the operation was of benefit. This was employed in 20 cases and only 2 of these or 10% showed any evidence of spasm after surgery.

The site of the aneurysm seemed to be of importance. Operation on those arising from the carotid siphon was the most likely to provoke spasm and treatment of those on the middle cerebral artery seemed the least dangerous in this respect. Other factors such as age, sex, pre-operative state, number of haemorrhages etc. details of which were given in the second paper, were considered but they appeared to have no influence on the development of spasm.



Fig 1 a) Pre operative angiogram aneurysm on posterior communicating artery no spasm
b) Post operative angiogram aneurysm occluded intense spasm of distal carotid siphon and of inferior part of pericallosal and middle cerebral arteries

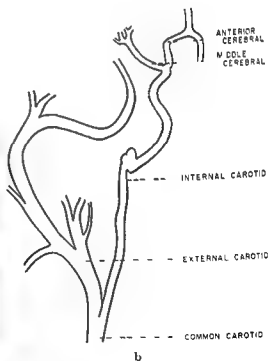


Fig 2 a) Normal anatomy of cranial arteries of dog showing common internal and external carotid and anterior and middle cerebral arteries b) Line drawing of right carotid system



Fig 3 a) Cont of film b) Narrowing of left internal and external carotid arteries and branches 1 min after injection of serotonin on left c) Normal appearances 8 min later

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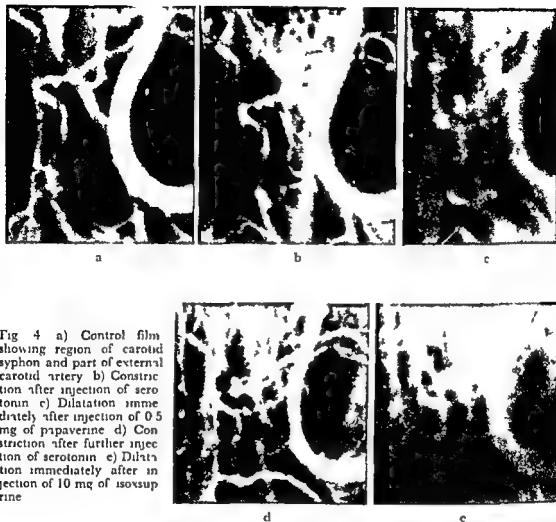


Fig 4 a) Control film showing region of carotid siphon and part of external carotid artery b) Constriction after injection of serotonin c) Dilatation immediately after injection of 0.5 mg of papaverine d) Constriction after further injection of serotonin e) Dilatation immediately after injection of 10 mg of isoxsuprine

There are many variables, so that it is hard to be sure that the figures quoted are of statistical significance. However, it is felt that they are of importance for the following reason. Of those that showed post operative spasm, only 46 % made a satisfactory recovery, without spasm the favourable outcome rose to 86 %.

First of all, the cause of the appearance of the vessels in the angiogram shown in Fig 1b should be determined. There are skeptics who think that the narrowing shown is due to low flow rather than to spasm. However, the blood in the vessels involved is in places flowing up vertically or around bends, and the arteries branch. Such circumstances would tend to break up any effect due to low flow. The irregular segmental distribution of the spasm that is often seen would also be against this theory.



Fig 5 a) Control film b) Constriction 1 min after injection of 1 mg of reserpine into artery

Others (e.g. TAVERAS 1963) have suggested that spasm occurs but that it is due to the contrast acting perhaps on an already irritable vessel. There are several points against this theory. It is not seen in other clinical states even when conditions favourable to the development of spasm might be thought to exist such as after operation on a vascular tumour. When spasm is present the contrast appears to be dammed back so that the same part of an artery is filled with contrast medium at a later stage than usual in a series of films. This suggests that the vessels were narrowed before the contrast ever reached them. Post operative spasm has been shown to be associated nearly always with clinical signs appropriate to the vessel or vessels involved (ALCOCK & DRAKE in publication).

Another possibility is that it may be due to the external pressure of a haematoma but the appearances sometimes seen with a large tumour or with cerebral oedema are quite different.

It seems reasonable therefore to accept that arterial spasm exists that it is not due to the contrast medium that it appears to be associated exclusively with subarachnoid haemorrhage and that it has a bad influence on the prognosis.

Clinically this problem has been approached here by delaying operation for 7 to 10 days after a haemorrhage unless recurrent haemorrhage or

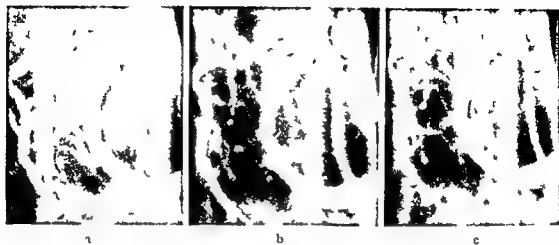


Fig 6 a) Control film b) No constriction after intra arterial injection of 1 mg of reserpine following treatment of dog with reserpine for 3 days c) Constriction still occurs after injection of serotonin

large haematomata make surgery imperative. During this time the patient is kept hypotensive to minimize the risk of further bleeding. At operation hypothermia and hyperventilation are avoided and the blood pressure is lowered still further. Angiography is carried out in the operating room to check on the obliteration of the sac and to see if spasm is present. After operation the blood pressure is raised to above normal with the hope of reducing the effect of any spasm if it develops, and this can be done safely with the knowledge that the sac is completely occluded. Alcohol is given in large doses if there is any suggestion of clinical spasm (HEWER 1963).

Since this regime was adopted, 30 patients have been seen with ruptured aneurysms. Three died awaiting operation. One of these was comatose, the other two would have been considered candidates for surgery. Seven either refused operation or were rejected for other reasons. Of the 20 that were operated on, 15 had post operative angiography. Only 2 of these, or 13%, showed evidence of spasm compared with 41% in the whole series. In both of these the constriction was only seen in the immediate angiograms and not in those done a week later. Only one patient who was operated on did badly, and at no time did she show any spasm.

It seems therefore that a small part of the problem has been solved by avoiding the factors mentioned and by more energetic post operative treatment if spasm does occur. However, it seems probable that these are merely precipitating agents and there is still some unknown underlying cause to be found. One that has been suggested (RAYNOR et coll 1961) is the release of serotonin from blood in the cerebro spinal fluid or from damaged brain.

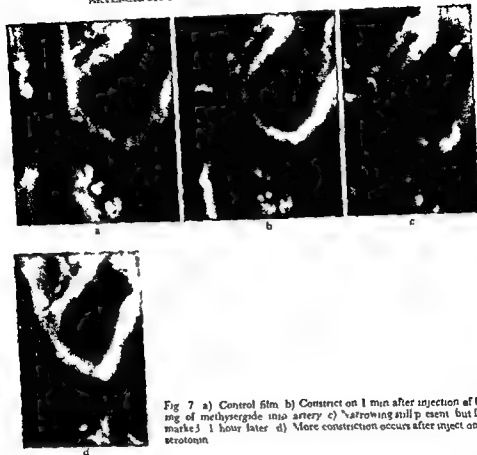


Fig 7 a) Control film b) Constriction 1 min after injection of 0.5 mg of methysergide into artery c) Narrowing still present but less marked 1 hour later d) More constriction occurs after injection of serotonin

With a view to studying these problems further a series of experiments on dogs has been undertaken. These involve the injection of contrast material usually 3 to 5 ml of Conray into one or both common carotid arteries and the size of the radiographic image is magnified approximately 5 times by using a 0.3 mm focal spot with a short anode-object and a long anode-film distance.

Fig 2 shows the normal anatomy which is somewhat confusing owing to the large and numerous branches of the external carotid artery. It is necessary to concentrate on a few of the larger arteries such as the internal carotid and anterior and middle cerebral vessels. On occasion there would be good contrast filling of the vertebro-basilar tree through the posterior communicating arteries and when only one side was injected there was usually filling of the arteries on the opposite side through the anterior communicating artery and other collateral channels.



Fig 8 a) Control film b) Constriction present 1 hour after intravenous injection of 1 mg of methysergide c) Further constriction after injection of serotonin

The effect of injection into the artery of small doses of serotonin was first tried. Fig 3a shows a control angiogram. In Fig 3b there is narrowing of the left internal and external carotid arteries and their branches one minute after the injection of 2 micrograms of serotonin on that side. Little if any change is seen on the right side, showing that the effect is local in nature. Fig 3c shows that the left side has returned to normal 8 minutes after the injection of serotonin.

A control film is shown in Fig 4a, while in Fig 4b the artery has been constricted by the injection of serotonin. Immediately after this, 0.5 mg of propranolol was injected into the artery, and this reversed the spasm and in fact dilated the vessels to well above their original calibre (Fig 4c). The markedly increased blood flow was demonstrated by the fact that the amount of contrast used had to be increased from 5 to 20 ml and even then the degree of filling of the arteries was not as good as previously. Narrowing occurred again (Fig 4d) after a further dose of serotonin. Isoxsuprine, 10 mg (KARLSBERG et coll 1963) was then injected into the artery, producing an effect similar to that after the administration of propranolol (Fig 4e).

It has already been stated that in the pre-operative period an attempt was made to keep the patient's blood pressure below normal. Reserpine has been considered as the hypotensive agent.

One effect of reserpine is to mobilize rapidly serotonin which has been stored in the cells. After a control film, injection into the artery of 1 mg of reserpine causes appreciable narrowing of the vessels one minute later (Fig 5). This may be due to the local release of serotonin although the author has not been able to prove it as yet.



FIG. 9 a) Control film b) Marked constriction of distal carotid siphon and of anterior and middle cerebral arteries following hyperventilation c) Dilatation of same vessels immediately after intra-arterial injection of 0.5 mg of papaverine

Another dog was given 1 mg of reserpine intramuscularly every day for three days in order to deplete his stores of serotonin. Injection now of reserpine into the artery no longer causes constriction (Fig 6b). This would tend to support the theory mentioned above and at least it shows that the solution itself causes no change in calibre. An amount of 5 micrograms of serotonin was then injected into the artery and the vessel was found to be still capable of reacting to it by constriction (Fig 6c).

These experiments would suggest that reserpine was perhaps not the ideal drug to use clinically under these circumstances as the initial release of serotonin might be just enough to precipitate spasm in a patient.

Methysergide is a derivative of lysergic acid and a direct antagonist of serotonin in its action on smooth muscle. This drug has been used in various experiments but so far the results have been disappointing. After the injection of 1/2 mg of Sansert into the artery there is in fact some constriction (Fig 7b) and this effect is still present although less marked 1 hour later (Fig 7c). Injection of 5 micrograms of serotonin at this time causes some further narrowing (Fig 7d).

This contraction of the arteries is also seen after the intravenous administration of methysergide. Fig 8 a and b show the appearances before and 1 hour after the injection of 1 mg of methysergide into a leg vein and in the second film there is definite constriction of the arteries. Injection now of serotonin again causes some further narrowing (Fig 8c).

Hyperventilation is known to cause constriction of the cerebral vessels (KRUEGER et coll 1963) (cf Fig 9). The initial arterial blood $p\text{CO}_2$ was 60 mm Hg and after hyperventilation and CO_2 absorption this fell to 13 mm Hg. In Fig 9b there is marked narrowing of the distal internal carotid and of the anterior and middle cerebral arteries and their branches. The external carotid artery and its branches are hardly affected at all, as is found in humans. This effect has been reversed, and the vessels are larger than initially, by the intra-arterial injection of 0.5 mg of papaverine (Fig 9c). There is again the increased blood flow with accompanying lowered concentration of contrast in the artery, which has only in part been corrected by the use of an increased volume of contrast.

The experiments that have been discussed are very incomplete and inconclusive, and it is recognized that much remains to be done before the ideas expressed have been proven correct or otherwise. In the majority of cases the doses used were relatively large and the injections made rapidly, and the experiments are being repeated with slower administration of smaller doses.

Most workers in this field have directed their attention towards the measurement of total cerebral blood flow or cerebrovascular resistance. With the use of such methods it is hard to know where any changes are taking place. It is felt that angiography offers a more direct and rational approach to the problem. It shows the part of the arterial tree that is affected, and remains the only method by which clinical arterial spasm can be confirmed.

Acknowledgements

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SUMMARY

Arterial spasm is a definite entity associated with subarachnoid haemorrhage and a potent source of post-operative morbidity. Its incidence can be reduced by delaying operation and by avoiding hypothermia and hyperventilation. In this preliminary investigation reserpine appears unsuitable as a pre-operative hypotensive agent and serotonin may be an underlying factor in the causation of clinical spasm. Methysergide did not prevent the spasm induced by serotonin.

ZUSAMMENFASSUNG

Arterieller Spasmus stellt ein einheitliches Bild bei Subarachnoidalblutung dar und kann die Ursache von postoperativen Komplikationen sein. Diese Gefahr kann durch Aufschieben von operativen Eingriffen und Vermeidung von Hypothermie und Hyperventilation ver-

mindert werden. Aus dieser vorläufigen Untersuchung geht hervor, dass Reserpin als präoperatives Hypotonicum ungeeignet ist und Serotonin ein ursächlicher Faktor beim Auftreten von Spasmus sein kann. Methysergid konnte den durch Serotonin ausgelosten Spasmus nicht verhindern.

RÉSUMÉ

Le spasme artériel est un état bien défini associé à l'hémorragie sous arachnoidienne et une source importante de complications post-opératoires. On peut réduire sa fréquence en retardant l'opération et en évitant l'hypothermie et l'hyperventilation. D'après cette étude préliminaire la réserpine ne paraît pas être un bon agent hypotenseur pré opératoire et la sérotonine peut être un facteur causal du spasme clinique. La méthysergide n'empêche pas le spasme dû à la sérotonine.

REFERENCES

- ALLCOCK J. M. and DRAKE C. G. Post operative angiography in cases of ruptured intracranial aneurysm. *J. Neurosurg.* 20 (1963) 752.
- — Ruptured intracranial aneurysms. The role of arterial spasm. *J. Neurosurg.* In publication.
- HEVER A. J. H. Symposium on anaesthesia for neurosurgery. *Brit. M. J.* 2 (1963) 240.
- KARLBERG I., ELLIOT H. W. and ADAMS J. E. Effect of various pharmacologic agents on cerebral arteries. *Neurology* 13 (1963) 772.
- KRUEGER T. P., ROCKOFF S. D., THOMAS L. J. and OMURA A. K. The effect of changes of end expiratory carbon dioxide on the normal cerebral angiogram. *Am. J. Roentgenol.* 90 (1963) 506.
- RAYNOR R. B., McMURTRY J. G. and POOL J. L. Cerebrovascular effects of topically applied serotonin in the cat. *Neurology* 11 (1961) 190.
- TAVERAS J. M. *Clinical Neurosurgery*. Williams & Wilkins, Baltimore, 1963, 292.

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CIRCULATION TIME AND PATTERN IN CEREBRAL ANGIOGRAPHY USING DIFFERENT TECHNIQUES FOR GENERAL ANAESTHESIA

by

A K AMUNDSEN, P AMUNDSEN and H RILSUM

The carbon dioxide tension of the arterial blood is regarded as the most potent factor influencing the cerebral blood flow. The normal limits for arterial $p\text{CO}_2$ are 35 to 45 mm of mercury. An admixture of 5 to 7 % carbon dioxide to the inspired air will, in normal persons, bring the $p\text{CO}_2$ up to roughly 50, and this has been shown to influence the cerebral circulation significantly. The blood flow, as determined for example by the nitrous oxide method, or by isotopes, increases and so does the circulation rate as determined during carotid angiography (SOKOLOFF 1959, KETY & SCHMIDT 1948, NOVAC et coll 1953, KRUEGER et coll 1963, TONNIS & SCHIEFER 1959, HARPER et coll 1961, LASSEN et coll 1963).

On the other hand it is a well known fact that actively conducted hyper-ventilation decreases cerebral blood flow and causes shrinkage of the brain, and it is therefore used as an effective method of reducing intracranial pressure during craniotomy.



Fig 1 Carotid angiography a) Relaxed with controlled respiration $p\text{CO}_2 = 40 \text{ mm Hg}$ b) Fluothane with spontaneous respiration $p\text{CO}_2 = 50 \text{ mm Hg}$



Fig 2 Left-sided carotid angiography in same patient as in fig 1 14 days later a) Fluothane with spontaneous respiration b) Fluothane with hyperventilation slow circulation and marked cross-circulation signs

Most drugs used for general anaesthesia tend to depress respiration and therefore may raise the carbon dioxide tension of the arterial blood. During the anaesthetic this is counteracted by controlled respiration, but during the period when spontaneous respiration is being resumed important changes in the carbon dioxide tension may be expected. Up to the present little attention seems to have been paid to the extent to which these variations may occur during routine anaesthesia and to whether under such circumstances alterations in cerebral

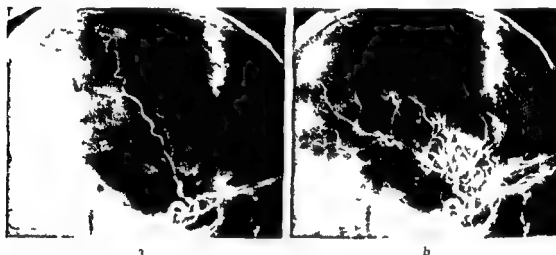
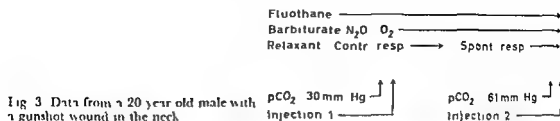


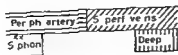
Fig 4 Carotid angiography a) Fluothane with controlled respiration pCO₂ = 30 mm Hg calibre of siphon 4 mm b) Fluothane with spontaneous respiration pCO₂ = 61 mm Hg calibre of siphon 5 mm

blood flow follow which may entail a risk of complications in patients with increased intracranial pressure

We have therefore studied the arterial pCO together with the cerebral circulation time and circulation pattern in carotid angiography, comparing different techniques for general anaesthesia in the same patient Care was taken to control and as far as possible to keep unchanged other factors such as blood pressure, pulse rate, injection time, position of catheter in the carotid artery and position of the patient

In one experiment general anaesthesia was started with barbiturate, nitrous oxide (N₂O) and oxygen, muscle relaxant and controlled respiration and during this period the pCO was 40 mm Hg in a blood specimen withdrawn immediately before injection 1 The anaesthetic technique was then changed The muscle relaxant was withdrawn so that spontaneous respiration recommenced and Fluothane was added Immediately before injection 2 the pCO₂ was 60 mm Hg Thus it is evident that the increase in pCO during

Injection 1
Fluothane
Spont resp
pCO₂ 51 mm Hg



Injection 2
Fluothane
Contr resp
pCO₂ 44 mm Hg

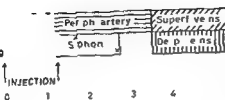


Fig 5 Circulation data from left side in a 63 year old female with a left sided central vascular lesion

routinely applied anaesthetic technique may be of the order which has been shown to influence cerebral blood flow significantly

Fig 1a shows a film from series 1 and Fig 1b a film from series 2 both exposed at the same interval from the start of injection. It is clearly seen that the speed of the cerebral circulation increased with elevated pCO₂ as did the calibre of the vessels

In the same patient a left sided carotid angiography was performed 14 days later. This time we had no pCO₂ determination but since the same technique was applied which had caused an increase at the first examination and since the circulation rate was obviously increased, a high arterial carbon dioxide tension probably was again present. Therefore the patient was actively ventilated for some minutes. Fig 2 shows one angiogram taken during spontaneous respiration which was depressed by the drugs, and another exposed at the same interval following injection but after active hyperventilation. Two interesting features noted were the marked slowing down of the circulation following hyperventilation and the marked cross circulation (No contralateral compression was applied). Probably this phenomenon must be attributed to the increased peripheral cerebral vascular resistance. HUBER (1964) has demonstrated the same phenomenon in cases of head trauma with severe oedema.

Fig 3 refers to another normal patient. Here the pCO₂ was subnormal during routine controlled respiration and without any change in the other drugs, the change over to spontaneous respiration produced an important increase

Fig 4a shows the decreased intracranial circulation at the time of the low pCO₂. The peripheral branches of the external carotid artery filled well and the siphon was narrow. Fig 4b is a comparably timed film when the carbon dioxide tension was 61. The external filling was less outstanding

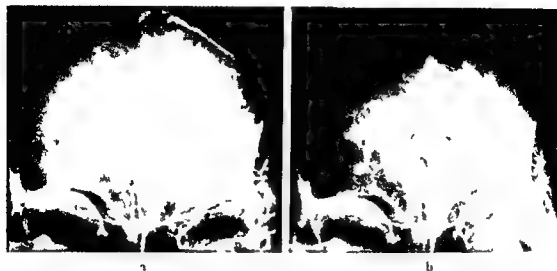


Fig. 6. Films from both series at 3.9 sec. a) Fluothane with spontaneous respiration $p\text{CO}_2 = 31$ mm Hg: superficial veins more evident. b) Fluothane with controlled respiration $p\text{CO}_2 = 44$ mm Hg: deep veins more evident.

and the internal circulation much quicker and the calibre of the siphon had definitely increased.

Fig. 5 is a diagram showing the circulation on the left side in a 65 year old female with a left sided central vascular lesion. In this case Fluothane and spontaneous respiration was applied in the first part of the examination and controlled respiration during the latter part. The increase of $p\text{CO}_2$ during spontaneous respiration was less than in the preceding cases but was still well beyond the normal upper limit, and within the range shown to influence the cerebral blood flow. During controlled respiration the $p\text{CO}_2$ was within normal limits.

The contrast disappeared more quickly from the arteries and appeared earlier in the veins in series 1 than in series 2 but in addition a further interesting observation was made. While the deep and superficial veins filled approximately simultaneously in series 2 with a normal $p\text{CO}_2$, the superficial veins filled definitely earlier and the deep ones later in series 1. The explanation may be that due to the deeply situated pathological process the adjacent part of the vascular bed could not respond adequately to the increased $p\text{CO}_2$, and therefore the blood was diverted through the superficial vessels in the unaffected areas.

A film from each series at 3.9 sec is shown in Fig. 6. With the increased $p\text{CO}_2$ the filling of the superficial veins was obviously dominant.

Conclusions

Our observations show that even during routinely conducted general anaesthesia rather wide variations in the $p\text{CO}_2$ may occur. These variations are followed by important changes in the cerebral circulation rate and there may also be alteration of the circulation pattern. Knowledge of these facts is important from a diagnostic point of view and it may be one explanation of the great spread of the normal circulation times during cerebral angiography given by different authors.

More important however is that such changes in the cerebral blood flow may cause deleterious effects in patients with increased intracranial pressure. In such cases a dilatation of the vessels may precipitate tentorial herniation. Certainly there exist cases where such complications have been attributed to the angiographic procedure instead of to the anaesthesia.

SUMMARY

Circulation time and circulation pattern in cerebral angiography have been studied comparing different techniques of general anaesthesia in the same patient. Very wide variations in the cerebral blood flow were observed. This is ascribed to the rather important variations in the arterial $p\text{CO}_2$ which may occur during routine anaesthesia.

ZUSAMMENFASSUNG

Es wurden die Zirkulationszeit und die Gefässe im cerebralen Angiogramm studiert, wobei verschiedene Technik von Allgemeinanästhesie am selben Patient miteinander verglichen wurde. Es wurden sehr grosse Variationen der cerebralen Blutzirkulation beobachtet. Dies wird den wichtigen Schwankungen des arteriellen $p\text{CO}_2$ die während Rutinanästhesie auftreten können zugeschrieben.

RÉSUMÉ

Les auteurs ont étudié le temps de circulation et le type de circulation en angiographie cérébrale en comparant différentes techniques d'anesthésie générale chez le même sujet. Ils ont observé de très grandes variations du débit sanguin cérébral. Ceci est attribué aux variations assez importantes de la $p\text{CO}_2$ artérielle qui peuvent se produire au cours de l'anesthésie ordinaire.

REFERENCES

- HARPER A. M., GLASS H. I., STEVEN J. L. and GRANAT A. H. The measurement of local blood flow in the cerebral cortex from the clearance of xenon. *J. Neurol. Neurosurg. Psychiat.* 27 (1964) 255.
- HUBER P. D. Bedeutung der Doppelfüllung bei der Karotisangiographie. *Fortschr. Röntgenstr.* 100 (1964) 114.

- KETY S. S. and SCHMIDT C. I. The effects of altered arterial tensions of carbon dioxide and oxygen on cerebral blood flow and cerebral oxygen consumption of normal young men. *J. Clin. Invest.* 27 (1948) 484.
- KRUGER T. P., ROCKOFF D., THOMAS L. J. and OMURA A. K. The effect of changes of end expiratory carbon dioxide tension on the normal cerebral angiogram. *Amer. J. Roentgenol.* 40 (1963) 506.
- LASSEN N. A., HOFDT RASMUSSEN K., SØRENSEN S. C. et coll. Regional cerebral blood flow in man determined by krypton 85. *Neurology* 13 (1963) 719.
- NOVACK P., SHENKIN A. K., BORTIN L. et coll. The effects of carbon dioxide inhalation upon the cerebral blood flow and cerebral oxygen consumption in vascular disease. *J. Clin. Invest.* 32 (1953), 696.
- SOKOLOFF L. The action of drugs on the cerebral circulation. *Pharmacol. Rev.* 11 (1959) 1.
- IONNIS W. und SCHIFFER W. *Zirkulationsstörungen des Gehirns im Serienangiogramm*. Springer Verlag Berlin 1959.

CONTROLLED TRIAL OF CERTAIN CONTRAST MEDIA IN CEREBRAL ANGIOGRAPHY

by

ARNOLD APPLEBY and GORDON L. GRYSPEERDT

With the increasing emphasis on the study of the smaller branches of the cerebral vessels for detailed diagnostic accuracy in cerebral angiography, the choice of the most suitable contrast medium has become even more important. Few workers have made any controlled comparison of the definition of the smaller cerebral arteries obtained with the numerous contrast media now available. The present investigation was planned to compare the angiographic quality of several different triiodo compounds containing the same amount of iodine per unit volume and the study was confined to percutaneous carotid angiography.

Method Employing the double injection method of stereoscopy in the lateral projection which we use as a routine in the investigation of cases with suspected space occupying lesions a different contrast medium was given for each injection of the stereoscopic pair. Alternate examinations received alternate media for the first injection.

Percutaneous puncture of the common carotid artery or internal carotid artery was performed using a No. 18 needle and 9 ml of the contrast medium

Table 1

Trial I — Comparison of Urografin 60% with Hypaque 45%

	Observer A	Observer B	Agreement between A and B
Urografin preferred	32	20	13
Hypaque preferred	4	1	0
No difference	30	41	25
Total cases	66	66	38
Significance of preference for Urografin	$p < 0.001$	$p < 0.005$	$p < 0.001$

at blood temperature were introduced at each injection. All examinations were carried out under general anesthesia which prevented any movement of the subject or displacement of the needle tip due to swallowing between injections. Any case in which readjustment of the needle was required, or in which there was alteration of the radiographic factors between the two series of exposures, was immediately excluded from the trial. No cases with proven subarachnoid haemorrhage or recent head injury were included in the investigation.

At the end of each trial, the relevant films of the cases were collected and displayed with all identification marks obscured, and two experienced observers separately compared the contrast density and definition of the small arteries in three pre-determined regions in each stereoscopic pair. The vessels chosen for this study were the anterior choroidal artery, the small branches of the callosomarginal artery and the small branches of the posterior parietal artery.

The preference, if any, for each group of vessels in one or other half of the stereoscopic pair was noted by each observer and his final preference in each individual case was determined by any preponderance in the detailed assessment. All the films were separately scrutinised before any of the identification marks were uncovered. The preferences of each observer were then tabulated and the results subjected to statistical analysis, both individually and combined.

Trial I

In the first trial Urografin 60% was compared with Hypaque 45%. Urografin 60% contains 52% N-methylglucamine diatrizoate and 8% sodium diatrizoate in aqueous solution, whereas Hypaque 45% contains only sodium diatrizoate in aqueous solution. Urografin 60% however, contains 29% of iodine per unit volume whereas Hypaque 45% contains only 27% of iodine per unit volume.

Table 2

Trial II — Comparison of Conray 60° with Angio-Conray 46.6°

	Observer A	Observer B	Agreement between A and B
Conray preferred	56	27	20
Angio-Conray preferred	8	9	2
No difference	37	63	30
Total cases	101	101	52
Significance of preference for Conray	$p < 0.001$	$p < 0.005$	$p < 0.001$

Results Observer A preferred Urografin in 32 angiograms Hypaque in 4 and found no difference in 30. Observer B preferred Urografin in 20 angiograms, Hypaque in 5 and found no difference in 41. The two observers agreed about the classification in 38 cases. In 25 there was considered to be no difference between the two contrast media and in 13 cases Urografin was considered to be superior. In no case was Hypaque preferred by both observers.

The differences between the two contrast media were extremely small in degree and it was in fact very difficult for either observer to detect any significant difference in many cases. In no case was it felt that the difference was such that the diagnostic interpretation of the cases examined was made easier with one contrast medium than with the other.

Statistical analysis The first observer found Urografin better in 32 cases and Hypaque better in 4 cases. These 36 cases alone are used for significance testing. On the null hypothesis that in cases in which a preference is expressed half will be in favour of each material the probability of this result is less than 1 in 10^4 . The result for this observer is thus highly significant.

The second observer found differences in 25 cases, preferring Urografin in 20 cases and Hypaque in 5 cases. The probability of this result is 5 in 10^4 . The result for this observer is thus also statistically significant.

If the analysis is confined to those cases in which both observers agreed as to preference it is found that agreement occurred in 38 cases of which there were 13 preferences for Urografin and none for Hypaque. The probability of this is 2 in 10^4 indicating a highly significant preference for Urografin (Table 1).

Trial II

In this trial in order to avoid a possible source of confusion the amount of iodine was exactly equalised in the two contrast media under test. The media chosen were Conray 60° and a specially prepared solution of Angio-Conray,

Table 3

Trial III — Comparison of Conray 61.7%, with Urografin 60%

	Observer A	Observer B	Agreement between A and B
Conray preferred	10	9	3
Urografin preferred	12	9	4
No difference	16	50	37
Total cases	68	68	14
Significance of preference	n.s.	n.s.	n.s.

46.6% both media containing 28% of iodine per unit volume. Conray is methylglucamine iothalamate in aqueous solution and Angio Conray is sodium iothalamate in aqueous solution.

Results. Again the differences in the quality of the angiograms were extremely slight, and in no case was it felt that the diagnostic interpretation was made easier with the one contrast medium than with the other.

Observer A preferred Conray in 56 angiograms and Angio Conray in 8 and found no difference in 37. Observer B preferred Conray in 27 angiograms, Angio Conray in 9 and found no difference in 65. The two observers agreed about the classification in 52 cases, in 20 of these Conray was preferred, in 30 there was no difference and in only 2 cases was Angio Conray preferred by both observers.

Statistical analysis of this trial showed a highly significant preference for Conray of the same order as that obtained for Urografin in Trial I (Table 2).

Trial III

In the third stage of the investigation it was decided to compare the preferred medium from Trial I with the preferred medium from Trial II. Urografin 60% (methylglucamine diatrizoate) was therefore compared with a specially prepared solution of Conray 61.7% (methylglucamine iothalamate) both media containing 29% of iodine per unit volume.

Although these two substances are isomeric, a toxic effect from their administration within a few seconds of each other could not be completely excluded. Experiments on anaesthetised cats however showed no alteration in the usual response to intra-arterial contrast media and it was therefore deemed safe to carry out the trial.

Results Observer A preferred Urografin in 12 angiograms Conray in 10 and found no difference in 46 Observer B preferred Urografin in 9 angiograms Conray in 9 and found no difference in 50 The two observers agreed about the classification in 44 cases in 37 of these there was no difference in 4 cases Urografin was preferred and in 3 cases Conray was preferred

Statistical analysis of this trial showed no significant preference for either contrast medium (Table 3)

Analysis of all trials

Four different contrast media were tested in three trials and any differences in definition and contrast density detected in the various media in the small cerebral arteries were found to be extremely slight in degree in all the trials This is shown by the number of cases in which the two observers disagreed and by the numerous cases in which no difference was detected by either observer (Tables 1 2 and 3)

In Trial I a highly significant preference for the medium containing methyl glucamine diatrizoate over the medium containing only sodium diatrizoate was found In Trial II there was a similar highly significant preference for the contrast medium containing methylglucamine iothalamate over the medium containing only sodium iothalamate

When the two media which were preferred in Trials I and II were tested against each other in solutions of equal iodine content in Trial III (one medium containing methylglucamine diatrizoate and the other containing methyl glucamine iothalamate) no statistical preference for either medium was found

Separate analyses of the results of the trials in relation to the 'up shift' and the down shift of the stereoscopic pairs and also in relation to the first and second injections irrespective of the contrast medium used consistently showed no statistical preferences

Discussion

There are surprisingly few reports in the literature of comparative studies of the angiographic quality of various contrast media carried out by controlled trials Most workers have combined an investigation of the toxic effects and the angiographic quality of various media by making comparison in separate series of subjects but a few have used double injection techniques in the same subject in which a controlled comparison has been possible The findings of the various authors concerning angiographic quality are difficult to assess as the results are usually expressed in general terms without statistical analysis and some conflict of opinion between authors is found

Table 4
Details of contrast media tested in the trials

Medium	Composition	Viscosity at 37.5 °C (in centipoise)
Conray 61 7 % (w/v)	Methylglucamine iothalamate	4.8
Conray 60 % (w/v)	Methylglucamine iothalamate	4.0
Urografin 60 % (w/v)	Methylglucamine diatrizoate 52 % + Sodium diatrizoate 8 %	4.0
Angio Conray 46.6 % (w/v)	Sodium iothalamate	2.2
Hypaque 45 % (w/v)	Sodium diatrizoate	2.1
Blood		2.7 (mean)

GROTE & BETTAG (1955) carried out clinical tests comparing Urografin for tolerance and contrast depth with Perabrodil M (methylglucamine 3,5 diiodo + pyridone N-acetate). These authors compared Perabrodil M 45 % with Urografin 60 %, on comparison of the contrast density of these concentrations, Urografin was found to be superior. The difference in contrast density of the two media on comparison of 60 % solutions of each was not so great but was still in favour of Urografin. However, GROTE & BETTAG in their series were comparing a diiodo compound with a triiodo compound.

DOCHNER & BRUGGER (1960) compared 50 angiograms using 60 % methylglucamine diatrizoate (Renografin), 50 angiograms using 50 % sodium acetrizoate (Urokon), and 10 angiograms using 50 % sodium diatrizoate (Hypaque). The arterial, capillary and venous phases of each examination were evaluated by a radiologist and a neurosurgeon separately. The density of the contrast medium in the angiograms obtained with sodium acetrizoate (Urokon) was found to be slightly less than that of those angiograms using methylglucamine diatrizoate (Renografin). When the small number of sodium diatrizoate angiograms was compared with the methylglucamine diatrizoate angiograms the density was found to be of equal degree in the arterial and capillary phases but methylglucamine diatrizoate was slightly superior in the venous phase. This superiority in the venous phase was felt by the authors to be due to the slightly higher viscosity of the methylglucamine salt resulting in slightly less dilution of the opaque medium.

A comparison was made between Isopaque 45 % (sodium metrizoate) and Hypaque 45 % in carotid and vertebral angiography by BULL & MURRAY

LESLIE (1962) In their carotid series these workers carried out successive injections first of one medium and then of the other into the same artery with only one puncture. Comparison of the carotid angiograms obtained with the two media revealed no difference in quality. These workers were, however, comparing two sodium salts.

KENDALL (1961) examined 100 patients undergoing carotid or vertebral angiography using Conray. In this series 50 of the patients received injections both of Conray 60% and of Hypaque 45% while this double injection was mainly carried out in order to test the degree of discomfort experienced by the patient: a direct comparison of the angiograms must have been possible in these 50 cases. KENDALL states that the contrast density achieved with Conray was excellent but no better than that obtained by using Urografin or Hypaque.

MARSHALL & LING (1963) as part of a very extensive clinical trial of Conray and Angio Conray in all types of angiographic procedures performed cerebral angiography using Conray 60% in 210 patients. These authors state that the contrast density was superior to that obtained with other contrast media.

A comparison of the quality of angiograms obtained with Hypaque 45% and Urografin 60% was carried out by BROADBRIDGE & LESLIE (1958). They found no significant difference in the level of contrast obtained with the two media but observed that Urografin was rather more viscous in the syringe.

In the present investigation we realised from our preliminary study of the angiograms and from the variation in the results of the comparative studies in the literature that the differences between the various media were very slight. Thus when we came to carry out the blind selection we were searching for extremely minor differences in contrast. This may account for our results differing from those of KENDALL (1961) and from those of BROADBRIDGE & LESLIE (1958). Our results agreed with those of MARSHALL & LING (1963) and with DOCHNER & BRUGGER (1960) in that we preferred the methylglucamine salts.

Solutions of methylglucamine salts are more viscous than solutions of the corresponding sodium salts and it is likely that the enhanced bolus effect of the more viscous medium is a major contributory factor to the better definition of the small arteries. The viscosities of the various contrast media in the concentrations employed in the trials are listed in Table 4.

Although the differences in the radiographic quality of the various contrast media detected by Trials I and II in the present investigation were very slight and the better media did not appear to enhance the diagnostic interpretation in the cases examined the finding of a highly significant preference for certain contrast media is important. The small vessels studied in this investigation were deliberately chosen as they are easily identifiable in all successful cerebral

angiograms by carotid angiography. However, some of the small central branches of the cerebral arteries may be difficult to identify in both carotid and vertebral angiography, especially in the antero posterior projections, even with the help of subtraction methods. The present investigation was therefore planned to determine the most suitable contrast medium for study of the small central vessels. If the viscosity of the contrast medium is a major factor in influencing the contrast density in these vessels it may be that the optimum value for this factor has yet to be found.

Since all the subjects in the three trials received injections of two media no attempt to compare the toxicity or side effects was made. However, no immediate unfavourable sequelae, such as a marked drop in blood pressure or apnoea, were encountered in any of the cases examined in this series.

Acknowledgments

We wish to thank Dr D. J. Newell of the Nuffield Department of Industrial Health, University of Newcastle upon Tyne, for the statistical analysis. We would also like to express our thanks to May and Baker Ltd for carrying out the animal experiments.

SUMMARY

A controlled comparison of the radiographic definition of the smaller arteries in cerebral angiograms obtained with certain contrast media has been carried out. A highly significant statistical preference for those media containing the methylglucamine salt over those containing the corresponding sodium salt was observed on careful scrutiny by two separate assessors. No statistical preference was found on comparison of two media containing the methylglucamine salts (Urografin and Conray) in solutions of equal iodine content.

ZUSAMMENFASSUNG

Es wurden die kleinen Arterien im cerebralen Angiogram bei Verwendung bestimmter Kontrastmittel miteinander verglichen. Kontrastmittel, die Methylglucaminsalze enthalten, sind denen mit dem entsprechenden Natriumsalz bei weitem vorzuziehen. Diese Beobachtung konnte von zwei voneinander unabhängigen Untersuchern gemacht werden und hat sich als statistisch hoch signifikant erwiesen. Eine statistisch gesicherte Überlegenheit eines zweier Kontrastmittel von Methylglucaminsalzen mit gleichem Jodgehalt (Urografin und Conray) konnte jedoch nicht festgestellt werden.

RÉSUMÉ

Les auteurs ont fait une comparaison contrôlée de la définition radiographique des plus fines artères sur les angiographies cérébrales faites avec certains moyens de contraste. L'examen soigneux par deux juges séparés a donné une préférence statistiquement très significative pour les moyens de contraste contenant le sel de méthylglucamine sur ceux contenant le sel de sodium correspondant. La comparaison de deux moyens de contraste contenant les sels de méthylglucamine en solutions de même teneur en iode (Urografin et Conray) n'a pas montré de préférence statistique.

REFERENCES

- BROADBRIDGE A T and LESLIE E V Cerebral angiographic contrast media Brit J Radiol 31 (1958) 556
- BULL J W II and MURRAY LESLIE R M Experience with metrizoate sodium (Isopaque) in cerebral angiography Farmakoterapi III (1967) 24
- DOCHNER G A and BRUGGER G E Comparison of diatrizoate methylglucamine sodium acetrizoate and diatrizoate sodium in cerebral angiography N Y St J Med 60 (1960) 4072
- GROTE W and BETTAG W Observations on the use of Urografin in cerebral angiography Fortschr Röntgenstr 83 (1955) 580
- KENDALL B E Cerebral angiography using Conray Brit J Radiol 37 (1964) 581
- MARSHALL T R and LING J T Clinical evaluation of two new contrast media Conray and Angio-Conray Amer J Roentgenol 89 (1963) 423

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conserve. L'hématome extra dural par contre d'après cet auteur est plus limité en étendue et sa forme sur les clichés de face est bi convexe la corticale cérébrale restant ainsi concave vers l'extérieur comme c'est le cas pour l'hématome sous dural chronique. Plus tard les travaux de HIRSCH, DAVID & BORNE (1962) ont permis de définir les éléments déjà connus du décollement angiographique de l'artère meningeuse moyenne dans le diagnostic d'un tel hématome extra dural. Plus récemment enfin les travaux de CROQVIST & KOHLER (1963) puis de HUBER (1964) ont permis d'établir avec plus d'exactitude les limites de nos possibilités diagnostiques en matière d'hématome extra dural.

Les précisions angiographiques que nous voudrions apporter en cas d'une telle affection reposent sur l'étude de nos 45 derniers cas d'hématomes extraduraux observés au service de Neuro Chirurgie de Colmar au cours de ces trois dernières années chez 42 malades. Ces précisions angiographiques portent sur des détails de l'image angiographique visibles sur les différentes phases de l'angiogramme : artérielle, veineuse et intermédiaire qu'il s'agisse de l'une de ces phases séparément ou sur plusieurs ou toutes ces phases.

Dans un grand nombre de nos hématomes extraduraux le diagnostic étiologique a ainsi pu être posé dans la période pré opératoire à l'aide des clichés et des incidences standard de face et de profil sans y adjoindre les projections spéciales en oblique par exemple. L'hématome extra dural étant une urgence chirurgicale le diagnostic doit pouvoir être posé rapidement et pour cette même raison les projections spéciales ne feraient qu'allonger l'examen. Certaines images angiographiques d'autre part surtout en cas de lésions bilatérales et multiples ne donnent que des signes discrets d'hématome qui sont l'objet essentiel de ce travail et qui n'inciteraient pas toujours le clinicien à pratiquer d'autres incidences.

Dans un premier chapitre assez concis nous présenterons nos cas d'hématomes extraduraux en tenant compte d'une part de leur localisation et d'autre part de la possibilité angiographique d'en faire le diagnostic étiologique.

Un deuxième chapitre sera réservé aux commentaires dans lesquels nous décrirons les images angiographiques qui nous paraissent caractéristiques d'un hématome extra dural.

Présentation et classification de nos hématomes

1. *Hématomes hauts et médians* Il s'agit de quatre cas de situation frontale empiétant vers l'arrière sur les parietaux. L'image angiographique est typique par le décollement du sinus longitudinal supérieur toujours visible sur les images de profil parfois sur les incidences de face lorsque le rayon central passe tangentiellement à la surface de l'hématome.

LE DIAGNOSTIC ANGIOGRAPHIQUE DE L'HEMATOME EXTRA-DURAL ET SES DIFFICULTES

par

J. BAUMGARTNER, I. WORINCER, J. P. BRAUN et D. LE MAISTRE

L'hématome extra dural est une affection traumatique dont la symptomatologie clinique est bien codifiée. La réalité cette symptomatologie classique ne se révèle pas toujours aussi exacte depuis que les cas de traumatisme crânien devenus plus fréquents sont centralisés dans les services spécialisés et donc mieux connus. Ainsi tel intervalle libre après traumatisme crânien a pu être attribué à un hématome intra parenchymateux ou sous dural aigu et un hématome extradural a pu être découvert chez un malade traumatisé crânien depuis quelques jours.

L'angiographie carotidienne examen routinier en cas de traumatisme crânien, a fait l'objet de nombreux travaux concernant l'hématome extra dural. Nous ne citons que les noms de LINDGREN (1951) qui disait que l'hématome extra dural ressemblait à l'hématome sous dural et ne s'individualisait que dans les localisations hautes et médianes par détachement du sinus longitudinal supérieur de la paroi crânienne. Cette notion ne confirmait d'ailleurs que celle déjà émise par WICKBOM (1949) et ENGESET (1950). NORMAN (1956) a fait un essai de différentiation entre l'hématome extra dural et celui de situation sous dural en montrant que leur forme et leur étendue n'étaient pas les mêmes. L'hématome sous dural aigu est plus étendu le long de la convexité cérébrale dont l'aspect convexe sur les clichés angiographiques de face reste

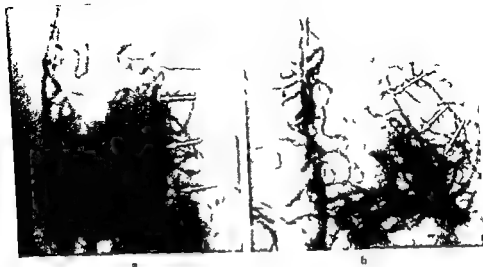


Fig 1 La ligne artérielle d'scont nue Il s'agit de modifications des vaisseaux artériels sylviens mais aussi de l'artère médiane du cerveau Plusieurs segments de ces vaisseaux se juxtaposent en une formation linéaire discontinue et curviligne dont la concavité extérieure épouse la membrane dure méningée refoulée par l'hématome L'étendue de ce dernier étant limitée les vaisseaux situés en avant et en arrière de lui peuvent atteindre la paroi crânienne et masquer le décollement artériel

a) Hématome extra dural de la convexité temporo-frontale b) Hématome extra-dural de la convexité pariétale c) Hématome extra dural de la convexité temporo-frontale Le décollement artériel est visible à la fin du stade artériel

et intermédiaire Si on ne dispose pas de ces cinq clichés il est parfois nécessaire de répéter l'examen afin d'obtenir tous les stades angiographiques et essentiellement les stades intermédiaires

II La ponction de l'artère carotide primitive doit être préférée à celle de l'artère carotide interne en cas de traumatisme crânien Elle permet l'opaci-

2 *Hématomes hauts et para médians* Il s'agit de ceux situés près de la ligne médiane sans avoir décollé le sinus longitudinal supérieur. Sur nos quatre cas de cette variété d'hématomes, trois étaient de situation frontale et un de localisation pariétale. Le diagnostic étiologique pré opératoire a été posé chez trois parmi eux sur les incidences de face et chez le quatrième (de situation frontale) sur les incidences de face et de profil.

3 *Hématomes de la base* Sur les neuf cas de cette variété d'hématomes, un était situé sous le lobe frontal et huit sous le lobe temporal. L'hématome frontal et trois hématomes de situation temporale présentaient un aspect angiographique extra durai typique. Les autres ne se différenciaient pas de l'hématome sous durai rigide ou intra parenchymateux. Un parmi les malades ayant présenté un tel hématome sous temporal était par ailleurs atteint d'un hématome hétéro latéral de la variété externe du paragraphe suivant.

4 *Hématomes de la convexité latérale de l'hémisphère cérébral* Ces hématomes ont en commun leur situation latérale au niveau de la région fronto-pariétalo-temporo-occipitale. Ils peuvent empiéter sur l'une ou l'autre de ces régions soit de l'avant vers l'arrière ou de la ligne médiane vers la base du crâne. Il s'agit de vingt-huit hématomes observés chez vingt-six malades. Deux parmi ces malades présentaient un hématome extra durai bilatéral de la variété latérale, un troisième malade présentait un hématome extra durai bilatéral dont un de la variété latérale et l'autre de la variété temporo-basale citée dans le paragraphe précédent. Vingt-six fois sur ces vingt-huit cas de cette variété externe l'hématome extra durai a été reconnu au stade pré opératoire. Parmi les deux cas non diagnostiqués avant l'intervention, une fois l'hématome a été visible angiographiquement mais malheureusement après que le diagnostic en fut posé à l'autopsie, le second de ces cas a été reconnu en cours d'intervention et n'a pas été visible angiographiquement.

Commentaires

Les réflexions qui découlent de l'étude angiographique de nos hématomes concernant quatre ordres de faits (1) la technique angiographique, (2) le contexte clinique, (3) la topographie de l'hématome, et (4) les signes angiographiques.

1. La technique angiographique

A. Afin de réunir avec beaucoup de régularité le maximum de signes angiographiques d'un hématome extra durai, il est utile de disposer d'une série graphique d'au moins cinq clichés de face et cinq clichés de profil. Cette série graphique permet d'englober tous les stades angiographiques artériels, veineux

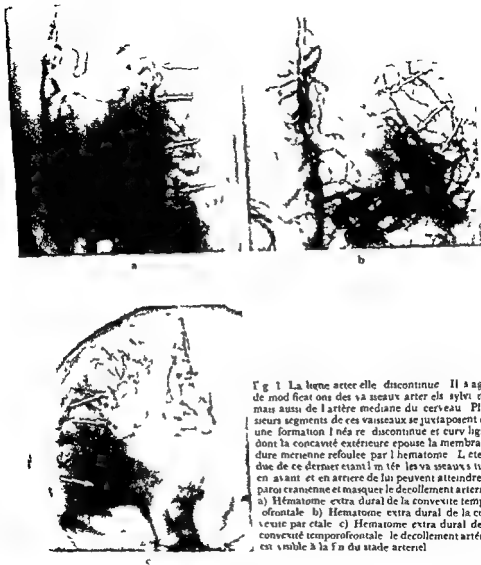


Fig 1 La ligne artérielle discontinue. Il s'agit de modifications des vaisseaux artériels sylvien mais aussi de l'artère médiane du cerveau. Plusieurs segments de ces vaisseaux se juxtaposent en une formation linéaire discontinue et curvilinear dont la concavité extérieure épouse la membrane dure méridienne refoulée par l'hématome. L'éten due de ce dernier étant limitée les vaisseaux situés en avant et en arrière de lui peuvent atteindre la paroi crânienne et masquer le décollement artériel.

a) Hématome extra dural de la convexité temporo-frontale. b) Hématome extra dural de la convexité parietale. c) Hématome extra dural de la convexité temporo-frontale: le décollement artériel est visible à la fin du stade artériel.

et intermédiaire. Si on ne dispose pas de ces cinq clichés il est parfois nécessaire de répéter l'examen afin d'obtenir tous les stades angiographiques et essentiellement les stades intermédiaires.

B La ponction de l'artère carotide primitive doit être préférée à celle de l'artère carotide interne en cas de traumatisme crânien. Elle permet l'opaci



Fig. 2 La ligne artérielle discontinue (sur une incidence de profil). Il s'agit d'un hématome pariétal paramédian. Dans cette variété d'hématomes le décollement artériel est toujours visible sur les incidences de profil parfois sur les incidences de face et de profil.

sification des branches de l'artère carotide interne et celles de l'artère carotide externe. Même si les extrémités distales de ces branches ne sont pas toujours reconnues individuellement en raison de la surcharge du cliché radiographique, elles peuvent contribuer parfois sur un petit segment de leur parcours à former avec les autres segments d'autres branches, un signe angiographique non négligeable d'un tel hématome extra-dural comme nous le verrons ultérieurement.

2. Le contexte clinique

L'interprétation correcte des angiographies exige la connaissance du fait clinique à savoir l'existence d'un traumatisme crânien relativement récent allant jusqu'à quelques jours. Cette notion est importante car certains signes angiographiques qui nous paraissent caractéristiques de l'hématome extra-dural ne le sont qu'à condition qu'il y ait cette notion de traumatisme récent. En effet ces signes peuvent exister en dehors de l'hématome extra-dural tel par exemple la ligne artérielle discontinue ou la ligne de condensation contrastée (ces signes seront explicités ultérieurement) qui peuvent se manifester dans d'autres circonstances. Tel est le cas par exemple de la ligne de condensation contrastée chez l'hématome sous-dural chronique.

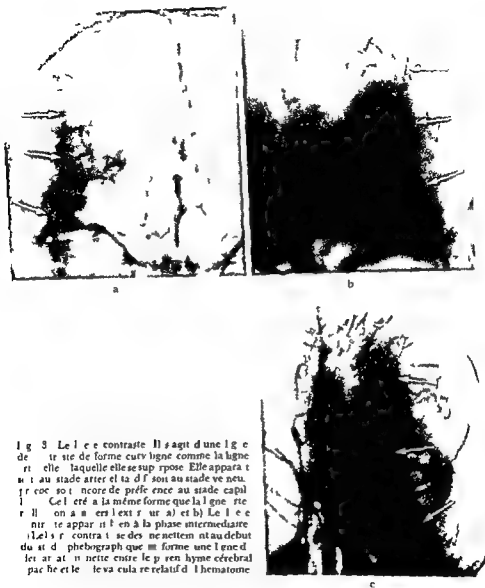


Fig 3 Le l e e contraste Il s agit d une l g e de tr ste de forme curv ligne comme la ligne rt elle laquelle elle se sup rpose Elle appara t m t au stade arter el ta d f son au stade ve neu pr coe so t ncore de p r l e nce au stade capil l Cel é r é a la même forme que la ligne ste r Il on a m ers l ext s ur a) et b) Le l e e ntr te appar t t en à la phase intermédiaire l e l s r contra t se des ne nettem nt au debut du st d phebograph que m forme une l gne d l e r at n nette entre le p ren hyme cérébral pac hie et le le va cula re relatif d l hematome

3 La topo,raphie de l'hematome

Cette topographie est importante a connaitre non seulement pour des raisons opératoires mais aussi et avant tout pour des causes diagnostiques En effect comme il ressort de l'étude de nos cas le diagnostic d hematome extra dural peut etre pose avec beaucoup de regularite et de certitude dans

certaines localisations qui sont (1) dans les variétés hautes et médianes par le refoulement du sinus longitudinal supérieur (2) dans les variétés hautes et para-médianes par un certain nombre de signes soit déjà connus et que nous rappellerons et certains autres que nous décrirons dans le paragraphe suivant, (3) dans les variétés latérales de toute la convexité cérébrale comme pour la variété précédente nous rappellerons ou décrirons les signes dans le paragraphe suivant.

Contrairement à ces trois variétés où l'hématome extra-dural peut être reconnu avec beaucoup de régularité, ceux de la base le sont moins souvent et ne peuvent, sauf à de rares exceptions près, être différenciés de l'hématome sous-dural ou intra-parenchymateux.

1. Les signes angiographiques proprement dits

Bien qu'il s'agisse de la partie la plus importante de ce travail, nous les citerons très brièvement. Ces signes ont pour certains déjà été décrits, pour d'autres nous paraissent nouveaux. Ils peuvent exister isolément ou simultanément soit sur le même cliché soit sur des clichés différents. On y distinguera ainsi selon les cas :

A. *La ligne artérielle discontinue* (Fig. 1 et Fig. 2). Il s'agit de modifications des vaisseaux artériels essentiellement du groupe sylvien mais aussi de l'artère parietallose. Ces modifications sont visibles sur les clichés de face pour la variété latérale des hématomes et de profil et parfois de face pour la variété des hématomes hauts et para-médians. Cette ligne artérielle discontinue est une formation artérielle linéaire formée par plusieurs segments artériels provenant de plusieurs branches de l'artère sylvienne ou de l'artère cérébrale antérieure et qui sont la cause de la discontinuité de cette ligne. Ces segments artériels donc se juxtaposent en prenant une forme linéaire discontinue et légèrement curviligne. Sur les clichés de face cette ligne artérielle présente une convexité extérieure correspondant à la dure-mère, qui subit la poussée de l'hématome. Sur les clichés de profil cette ligne est concave vers le haut et vers l'arrière pour les hématomes para-médians postérieurs et plutôt concave vers le haut et vers l'avant pour les hématomes para-médians antérieurs. La formation de cette ligne artérielle discontinue est certainement favorisée par le refoulement régulier et curviligne de la membrane dure maternelle par l'hématome.

B. Le second signe de l'hématome extra-dural est également un signe artériel puisqu'il s'agit du *refoulement de l'artère méningée moyenne*. Ce signe a déjà été décrit. Notons simplement que l'artère méningée moyenne ne peut pas toujours être individualisée et que lorsqu'elle est présente elle n'est pas toujours intéressée par le refoulement de l'hématome. Il s'agit donc d'un signe ayant une valeur pathognomonique lorsqu'il est présent mais qui est



Fig. 4 Refoulement des veines corticales en regard de l'hématome extra dural qui se traduit par ailleurs par un \downarrow de vascularisation relatif

malheureusement fréquemment absent et devient ainsi de par son inconstance un signe moins intéressant

C Le *vide vasculaire relatif* (Fig 3 c et Fig 4) apparaît fréquemment au cours de la phase capillaire ou intermédiaire à l'endroit même où siège l'hématome. Ce *vide relatif* peut contraster par une ligne de démarcation plus ou moins nette avec le parenchyme cérébral qui présente une meilleure opacification

D A cette ligne de démarcation correspond dans un bon nombre de cas la *ligne de condensation contrastée* (Fig 3). Celle-ci apparaît souvent soit à la fin du stade artériel soit au cours de la phase intermédiaire ou aucune phase ni artérielle ni veineuse n'est véritablement reconnue. Cette ligne de condensation est parfois également visible sur d'autres clichés de la sertiographie. Elle est habituellement en rapport avec la *surprojection tangentielle* de plusieurs éléments vasculaires opacifiés tel le stade terminal de la ligne artérielle discontinue, la démarcation entre le *vide vasculaire relatif* et le parenchyme cérébral mieux opacifié l'artère méningée moyenne sur un de ses segments même si elle n'est pas bien individualisée ou encore certains éléments veineux. Elle est caractéristique de l'hématome extra dural à condition qu'il existe la notion de traumatisme crânien récent. Cette ligne est comparable et ressemble à celle qui sépare en cas d'hématome sous dural chronique l'hématome de la convexité cérébrale.

F Au stade veineux un *refoulement d'une veine corticale* (Fig 4) est parfois visible en regard de cette ligne de démarcation entre hématome et parenchyme

cerebral. Ce refoulement est moins caractéristique et ne permet pas à lui seul le diagnostic d'hématome.

F. Une *extravasation du produit de contraste* dans la poche hématique. Il s'agit d'un signe déjà bien connu et caractéristique. C'est cette extravasation du produit de contraste qui permet parfois de poser avec certitude le diagnostic d'hématome extra-dural lorsque ce dernier a un siège basal.

G. Le dernier signe et le premier à avoir été décrit. Il s'agit du *décollement du sinus longitudinal supérieur* de la paroi crânienne, en cas d'hématomes hauts et médians.

Il est utile de connaître tous ces signes qui peuvent contribuer au diagnostic d'hématome extra-dural. Ces signes peuvent exister isolément ou simultanément même dans les cas où aucun décollement des vaisseaux de la paroi crânienne n'est visible et donc dans les cas où il n'existe pas de vide vasculaire du à l'hématome. Cette éventualité n'est pas rare en cas de lésions cérébrales bilatérales.

RÉSUMÉ

Sur une série de 45 examens angiographiques d'hématomes extra-duraux, les auteurs ont pu reconnaître régulièrement les hématomes médians qui décollent le sinus longitudinal supérieur de la paroi crânienne, les hématomes para-médians et les hématomes latéraux de la convexité cérébrale. Ces deux dernières variétés présentent des signes caractéristiques qui sont la ligne artérielle discontinue et la ligne de condensation contrastée, auxquels s'ajoutent parfois d'autres signes déjà connus. Les hématomes extra-duraux de la base ne peuvent généralement pas être distingués par l'angiographie des hématomes sous-duraux ou intra-parenchymateux.

SUMMARY

In a series of 45 angiographic examinations of extradural hematomas the authors regularly recognized three types: median hematomas that separate the superior longitudinal sinus from the cranial wall, paramedian hematomas, and lateral hematomas of the cerebral convexity. The last two varieties produce characteristic signs including discontinuation in the arterial course and density of contrast medium. Other signs already known are also occasionally seen. Extradural basal hematomas cannot as a rule be distinguished by angiography from subdural or intracerebral hematomas.

ZUSAMMENFASSUNG

In einer Serie von 45 angiographischen Untersuchungen extraduraler Hämatome haben die Verfasser regelmässig drei Typen erkennen können: mediane Hämatome, die den oberen longitudinalen Sinus von der Schädelswand trennen, paramediane Hämatome und laterale Hämatome über die Konvexität. Die letzten zwei Varianten geben charakteristische Zeichen, so wie Unterbrechung der Arterien und Veränderung der Kontrastmitteldichte. Andere bereits bekannte Zeichen sind auch von Zeit zu Zeit zu sehen. Extradurale basale Hämatome können in der Regel nicht von subduralen oder intraparenchymatösen Hämatomen mittels Angiographie unterschieden werden.

REFERENCES

- CROONIS S and KOHLER R : Angiography in epidural haematomas Acta radiol D 1 (1963) 42
- ENGLEST A : On roentgen examination in head trauma Acta radiol 34 (1950) 288
- HIERCH J F, DAVID M et BORNE G : Un signe angiographique des hématomas extra-duraux Neuro-chirurgie 5 (1967) 91
- HILBER P : Die Zerebrale Angiographie beim frischen Schadel Hirn Trauma Thieme Verlag Stuttgart 1964
- LINDEREN E : Roentgenologie in Handbuch der Neurochirurgie Bd 2 Edit von Olivecrona und Tonnies Springer Berlin 1954
- NORMAN O : Angiographic differentiation between acute and chronic subdural and extra dural haematomas Acta radiol 46 (1956) 371
- WICKBOM J : Angiography in post traumatic intracranial hemorrhage Acta radiol 32 (1949) 749

ANGIOGRAPHY FOR EXTRACRANIAL TUMORS

by

ARNOLD BERRETT

We have found angiography to have many important and practical applications in the management of certain extracranial tumors (BERRETT 1963). The indications may be discussed under three headings: pre-operative, post-operative, and regional infusion techniques (intra-arterial).

Pre-operative indications

Mass lesions of the head and neck of uncertain origin Angiography is of value in demonstrating the feeding vessels, degree of vascularity and anatomical extent of the tumor. In certain cases it may even aid in the diagnosis. A few tumors have a characteristic angiographic appearance, for example carotid body tumors, but others show little or no deviation from the normal. As a general rule, in a given tumor the vascular areas are the more malignant. This may indicate the best site for biopsy.

Vascular occlusion Any large mass situated on the lateral aspect of the neck may encircle and compress the carotid artery causing various degrees of occlusion. In such cases it is frequently difficult to determine clinically whether the carotid artery is patent or not. An example of this is illustrated in Fig. 1.

Lesions of the vault or base of the skull with possible intracranial extensions Occur



Fig 1 B a) Anteroposterior (A/P) view. The commencement of the carotid artery is normal but more distally the vessel is compressed by an extensive mass from a carcinoma of the right tonsil. b) Lateral view. A narrow threadlike segment of the internal carotid artery is seen. More distally the lumen becomes wider and more normal in caliber.

sionally one is presented with a soft tissue mass of the scalp which is associated with underlying bony destruction of the vault. In such cases it may be difficult to determine whether there is in addition intracranial but extradural extension. If intracranial extension does take place the angiographic appearance will depend largely on its location. For example an osteolytic metastatic deposit in the midline of the vault which is associated with extradural extension will displace the superior sagittal sinus away from the inner table of the skull (Fig. 2).

Post operative indications

Trauma to the carotid artery leading to vascular occlusion. An example of this is seen in the following case. The patient was a 44 year old male who presented with a slow growing right sided mass on the lateral aspect of the neck. At

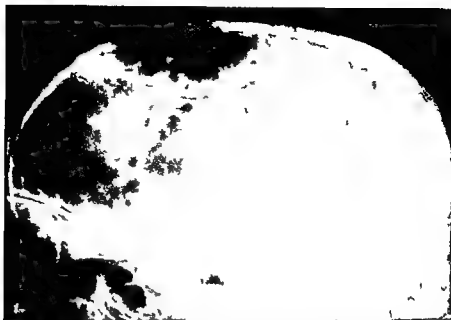


Fig. 2 Angiogram in late arterial phase showing metastasis from a hypernephroma with destruction in the vault of the skull and extradural extension. The small arteries form a vascular halo around the area of extradural extension.

surgery this mass was adherent to the carotid artery and very difficult to excise. Histological examination revealed it to be an extracranial meningioma. The patient had a stormy post operative course with features of an acute and severe cerebral vascular accident on the left hand side. This ultimately led to his death. Utilizing the clinical, angiographic and post mortem information at our disposal, the following appears to have taken place. The carotid artery in the neck was traumatized at the time of surgery, and this resulted in subintimal hemorrhage associated with thrombosis in the lumen. This led to vascular occlusion and extensive infarction of the right side of the brain.

A very careful post mortem examination failed to reveal an associated meningioma of the nervous system. We therefore regard this as an example of an extremely rare meningioma occurring completely outside the nervous system.

Complications in patients with known carcinoma. Some of our patients with known primary carcinoma presented with neurologic findings in the post operative period. We have found angiography to be of very great value for the confirmation of metastatic deposits and for the demonstration of other unrelated intracranial diseases presenting a similar picture (BERRETT 1963).

Regional infusion techniques (intra arterial)

This is now employed in several centers for protracted and continuous infusion of various chemotherapeutic agents and radioactive isotopes to the tumor bearing area. It is of the utmost importance to select the most appropriate artery prior to this form of therapy and essential that the catheter is correctly placed and maintained in position (Horowitz 1960)

SUMMARY

The full demonstration of both the normal and pathological arterial systems of fundamental importance in the correct handling of many extracranial tumors. Specific indications in both the pre and post-operative period are described.

ZUSAMMENFASSUNG

Die vollständige Darstellung des normalen und pathologischen arteriellen Gefäßsystems ist für die richtige Behandlung bei vielen extrakraniellen Tumoren von fundamentaler Bedeutung. Spezifische Indikationen in der prä- und postoperativen Periode werden beschrieben.

RÉSUMÉ

Il est d'une importance fondamentale de faire un examen complet des artères normales et pathologiques pour traiter correctement un grand nombre de tumeurs extracrâniennes. L'auteur décrit les indications spécifiques de l'angiographie dans les périodes pré et post opératoires.

REFERENCES

- BERRETT A. Head and neck arteriography. Trans Amer Acad Ophthal Otolaryng 67 (1963) 530
- Metastatic disease in the brain and its differential diagnosis. Acta radiol 1 Diagnosis (1963) 206
- LOCKHOTT W. P. and EVANS K. T. The place of soft tissue arteriography. Brit J Radiol 37 (1964) 167
- HAWKINS T. D. Glomus jugulare and carotid body tumors. Clin Radiol 12 (1961) 199
- HOROWITZ H. Intra arterial therapy: a review of its clinical application in malignant disease. Brit J Radiol 33 (1960) 659

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HIGH SPEED CINEFLUOROGRAPHY OF CERVICOCEREBRAL BLOOD FLOW PATTERNS IN DOGS

by

A BONAKDARPOUR, P R LANCH, T MURTACH and H M STAUFFER

Since the introduction of cerebral angiography by MONIZ (1927) and rapid serial angiography by CURTIS (1949) considerable data has been collected on the roentgen anatomy of both intracranial and extracranial vessels. Little radiologic physiologic information about the cervicocerebral circulation is available, however. Since the invention of electronic x-ray image intensifiers by COLTMAN (1948), the technique of cinefluorography (BONAKDARPOUR 1958) has been applied to investigate the physiology of different organs and especially the cardiovascular system (STAUFFER et coll 1957). With the improvement of image amplifiers, high speed cinefluorography has become available. A technique of precision motion analysis of high speed cinefluorograms of particulate contrast medium injected into the blood vessels of living animals has been developed in the radiology physiology laboratory of the Temple University Medical Center. This method has been used by several investigators for the study of blood flow in different vessels. LANCH et coll (1960) studied the flow patterns in the venous system in cats. GIMENEZ and collaborators (1961) reported on analysis of the flow patterns of particulate contrast material



Fig. 1. Angiography by injection of Renografin 60 into the brachiocephalic artery of dog. Right carotid, left carotid and right vertebral arteries originate from the brachiocephalic artery.

in blood vessels. OHLSSON (1962) investigated the left heart and aortic blood flow in the dog using this technique. PETERSEN studied the blood flow patterns of the distal thoracic and abdominal aorta in dogs. We have used this method for the study of normal cervicocerebral flow in dogs for the establishment of a base line and this data will be applicable to studies of abnormal cervicocerebral flow.

Methods. A total of 44 mongrel dogs weighing from 5.5 to 23 kg were used. The animals were anesthetized by the intravenous injection of 32.5 mg Pento-barbital sodium per kg. In our first 14 experiments the right carotid and left vertebral arteries were studied separately in each animal; in the last 33 experiments the carotid and vertebral arteries were studied simultaneously. Two red Ödman catheters were introduced, one in each femoral artery. One catheter was advanced into the brachiocephalic artery from which originate the right carotid, the left carotid and the right vertebral arteries (Fig. 1). The other catheter was used for the recording of blood pressure from the distal abdominal aorta. The ECG, heart sounds and blood pressure were constantly

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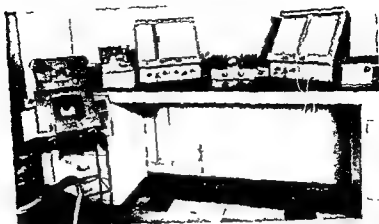


Fig. 3 Data plotting equipment (from left to right) Vanguard motion analyzer, calibrating control panel, X-Y recorder (Electro Instruments Inc.), Philbrick four-pole analog computer, X-Y recorder with curve follower (Electro Instruments Inc.) and power supply.

Table

Dog No.	Body weight (kg)	Angiographic vessel diameter (mm)	Electromagnetic flow rate (ml/sec)		Electromagnetic flow velocities (cm/sec)		H S cinefluorography plotted velocities (cm/sec)		Associated artery
			Max.	Min.	Max.	Min.	Max.	Min.	
37	17.7	8.9	125	374	91.9	6.95	20.0	6.00	Vertebral
3	17.7	3.01	3.66	1.64	50.6	27.8	57.0*	30.0	Carotid
35	21.0	2.14	1.00	0.77	8.0	2.14	37.0	9.60	Vertebral
35	21.0	3.34	4.97	9.60	56.6	29.7	56.0	15.0	Carotid
39	16.4	2.34	3.9	4.67	74.5	10.9	70.0	6.68	Carotid
47	15.6	9.27	3.93	1.33	79.8	37.8	84.4	41.8	Carotid
4	15.6	2.00	1.16	217	36.9	6.9	34	6.98	Vertebral
44	18.7	9.50	5.9	1.34	120	27.4	91.0	29.2	Carotid
44	18	2.60	6.28	6.5	118	17.3	80.5	19.9	Carotid
4	9.3	.50	4.84	834	98.8	17.0	84.6	96.0	Carotid
46	21.3	4.6	4.41	1.33	93.0	28.0	140	38.2	Carotid

These figures represent the maximum velocity at the time of flow meter and high speed cinefluorography comparison studies. However, maximum carotid velocity was 90 cm/sec in this dog by the droplet technique at the beginning of the study and decreased to 57 cm/sec after a total injection of 2.3 ml of Ethiodol by the same technique.

Droplet time-displacement curve was plotted near the area of the flow meter probe.

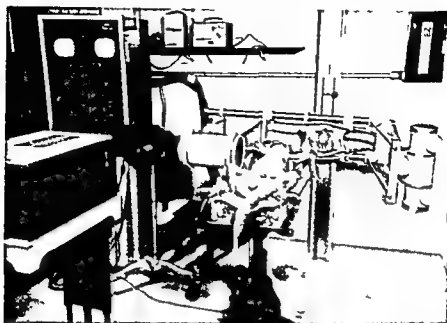


Fig 2 Equipment used for high speed cinefluorography high speed movie camera a six inch Philips image amplifier the electronics for medicine multichannel oscilloscope and recorder a Tektronix 502 dual beam oscilloscope and roentgen ray equipment

monitored on an electronics for medicine multichannel recorder (Fig 2), and recorded when appropriate. High speed cinefluorograms of the lateral view of the neck were made during the injection of 0.1 to 0.5 ml Ethiodol (iodized oil from E. Fongera and Co Inc., Hicksville, New York) into the brachiocephalic artery. With proper localization of the tip of the catheter in the brachiocephalic artery, the droplets of Ethiodol entered both carotids and the right vertebral artery. The heart sounds and blood pressure were also recorded on the high speed movie film from a Tektronix 502 dual beam oscilloscope through a side lens of the high speed camera simultaneously with the fluoroscopic image (GIMENEZ et coll 1961, LYNCH et coll 1960, OHLSSON 1962). In each experiment, cineangiography of the cervicocerebral region was performed by the injection of Renografin 60% (diatrizoate methylglucamine from E. R. Squibb & Sons, New York) for the measurement of vessel diameter.

The high speed cinefluorograms were analyzed on a Vanguard motion analyzer (Fig 3). Time displacement curves of the droplets as well as blood pressure and heart sound recordings were plotted on an X-Y plotter, and velocity and acceleration curves were then plotted from the time displacement curves by the use of a curve follower on an X-Y plotter (GIMENEZ et coll 1961).

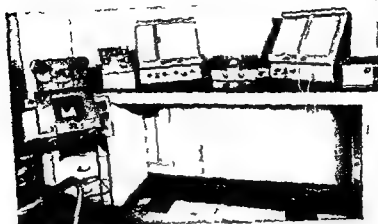


Fig. 3 Data plotting equipment (from left to right): Vanguard motion analyzer, calibrating control panel, XY recorder (Electro Instruments Inc.), Philbrick four-ply analog computer, XY recorder with curve follower (Electro Instruments Inc.) and power supply.

Table

Dog No.	Body weight (kg)	Ago-graphic vessel diameter (mm)	Electromagnetic flow rate (ml/sec)		Electromagnetic flow velocities (cm/sec)		H S cinefluorography plotter velocities (cm/sec)		Associated artery
			Max	Min	Max	Min	Max	Min	
3	17.7	2.87	1.25	.374	21.2	6.23	20.0	6.00	Vertebral
3	17.7	3.04	3.66	1.64	50.6	22.8	52.0	30.0	Carotid
35	21.0	2.14	1.00	.077	78.0	2.14	32.0	2.60	Vertebral
35	21.0	3.34	4.97	2.60	56.6	29.7	56.0	15.0	Carotid
39	16.4	2.33	3.2	.467	74.5	10.9	70.0	6.68	Carotid
47	15.6	2.27	3.23	1.33	79.8	3.8	84.4	41.8	Carotid
4	15.6	2.00	1.16	.717	36.9	6.9	31.2	6.28	Vertebral
44	18.7	2.50	5.97	1.34	120	77.4	91.0	79.2	Carotid
44	18.2	2.60	6.78	6.5	119	12.3	80.5	19.9	Carotid
45	3	2.50	4.81	.834	98.8	17.0	81.6	75.0	Carotid
46	21.3	2.46	4.41	1.33	93.0	28.0	140*	38.2	Carotid

* These figures represent the maximum velocity at the time of flow meter and high speed cinefluorographic comparative studies. However, maximum carotid velocity was 90 cm/sec in this dog by the droplet technique at the beginning of the study and decreased to 57 cm/sec after a total injection (2.3 ml) of Ethiodol by the same technique.

Droplet time-displacement curve was plotted near the area of the flow meter probe.

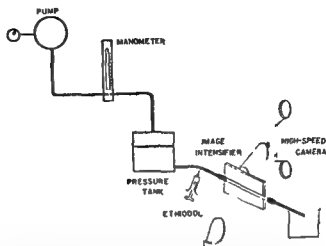


Fig 4 Schematics of procedure for hydrodynamic model experiments Fisher pressure vacuum single shaft air pump mercury manometer constant pressure reservoir calibrated collection beaker

When the velocities in the carotid artery were very high, the actual velocity had to be calculated from the time displacement curves using the formula $\bar{V} = \frac{\Delta x}{\Delta t}$ where Δx is the displacement of the droplet and Δt is the time (SEARS &

ZEMANSKY 1963) Accuracy of the Vanguard motion analyzer technique for converting time displacement curves to velocity measurements decreases as the velocity increases. Thus direct geometric measurements and calculations must be performed on high velocity droplet movement, so that there is no actual decrease in total accuracy of the method.

Two experiments were performed with a dog in the prone position.

In 11 experiments an electromagnetic flow meter was used for recording the blood flow. Heart sounds (or blood pressure) and flow were simultaneously recorded on the high speed cinefluorograms during the injection of Ethiodol droplets (see Table).

For the further evaluation of this droplet technique, two series of hydrodynamic model experiments have been performed (Fig 4) using steady flow.

In the first group of model experiments volume flow of water was measured through a siliconized polyethylene tube with an internal diameter of 1.778 mm under constant pressure. By knowing the flow rate, the average velocities (SEARS & ZEMANSKY 1963) through the tube with and without injection of Ethiodol droplets were calculated. In the second group of model experiments, the average velocity was measured by simultaneous volumetric and high speed cinefluorographic techniques.

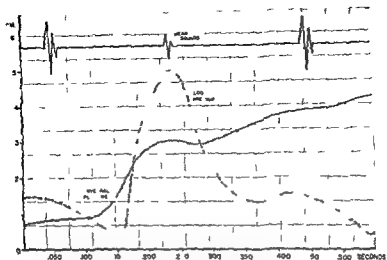


Fig. 5. A time and displacement recording of Ethiodol droplet movements from vertebral artery analysis. The X axis represents the time in seconds and the Y axis the displacement in centimeters along the vessel.

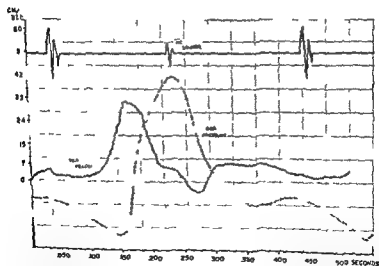


Fig. 6. Velocity curve of vertebral artery droplets (same artery as shown in fig. 5). Note the time lag of the rise of blood pressure in the distal aorta and velocity changes in the vertebral artery. This is also demonstrated in fig. 5. The X axis represents the time in seconds and the Y axis the velocity in centimeters per second along the vessel.

Results

Comparative velocities The velocity of droplet flow in the common carotid is higher than that of the vertebral artery. The maximum instantaneous (SEARS & ZEMANSKY 1963) systolic velocity in the common carotid artery ranged between 60 and 107 cm/sec. The maximum instantaneous systolic velocity in the vertebral artery is always lower and varies from 30 to 60 % of that of the common carotid artery. An example of a time displacement curve of droplet movement through the vertebral artery with simultaneous blood pressure and heart sound tracings is shown in Fig 5. The X axis reveals the time which has been calculated from the number of frames per second (each frame is 3.7 milliseconds). The Y axis shows the displacement of a droplet in centimeters after the correction for geometric magnification. The velocity curve (maximum 32 cm/sec) recorded from the same vertebral artery is seen in Fig 6. The maximum instantaneous systolic velocity in the common carotid artery of this dog was 90 cm/sec. A time displacement curve of droplet movements in the common carotid artery in another dog is shown in Fig 7 and the velocity curve (maximum 93 cm/sec) of the same common carotid artery in Fig 8.

Fig 9 shows an example of the velocity curve of droplets passing through the vertebral artery as compared with a curve of blood flow passing through the same artery measured by an electromagnetic flow meter and recorded by high speed cinefluorography. The flow curve has been analyzed by our technique and the corresponding velocities are shown on the ordinate at the level of the flow curve. The two curves are quite similar in shape and amplitude; the phase shift is due to the time rise of the flow meter. Fig 10 illustrates the same curves with the common carotid velocity curve (by droplet technique) superimposed in order to show the comparative velocities by the techniques and also to compare the velocity of the common carotid and vertebral arteries.

Model experiments In 34 separate model experiments the average velocity with Ethiodol was 174 cm/sec and without Ethiodol 189 cm/sec with an 8 % possible error. In nine model experiments comparing the volumetric and high speed cinefluorographic methods the measurements confirmed the reliability of the latter technique with a difference in average velocities of only 17 cm/sec in a range of 137 cm/sec to 173 cm/sec.

Back flow in the cervicocerebral vessels No back flow was observed within the common carotid artery of an anesthetized normotensive animal with the initial injection of the radiopaque particulate contrast medium. However back flow was observed in the vertebral artery of normotensive animals



Fig. 7 The displacement recording of catheter tip analysis from the carotid artery. The time lag has been corrected for so that the curves are tracing by plotting the curve to the left. The X axis represents the time in seconds and the Y axis the displacement in centimeters along the vessel.

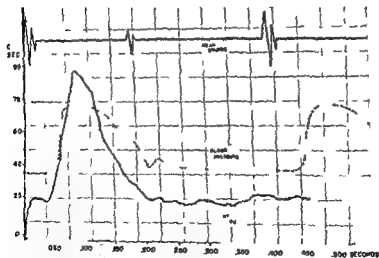


Fig. 8 Velocity curve of the analyzed carotid artery droplet (same artery as shown in Fig. 7). The time lag has been again corrected for in the displacement tracing by shifting the curve to the left. The X axis represents the time in seconds and the Y axis the velocity in centimeters per second along the vessel.

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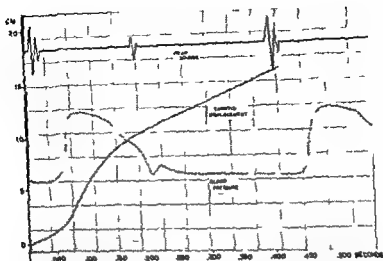


Fig 7 Time displacement recording of 81h. dol droplet analysis from the carotid artery. The time lag has been corrected for in the blood pressure tracing by displacing the curve to the left. The X axis represents the time in seconds and the Y axis the displacement in centimeters along the vessel.

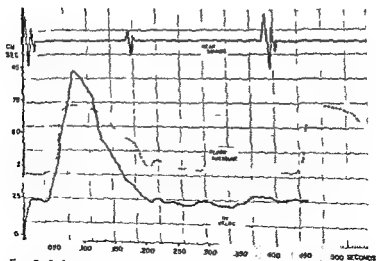


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(377 ml/min) is lower than the highest mean flow rate of the human internal carotid artery (494 ml/min) as reported by HARDESTY et coll (1960)

Velocity of blood but not flow may change directly under the flow probe of the electromagnetic flow meter if the vessel size (external diameter) is larger than the internal diameter of the probe. Variations in vessel size are impossible to control so that occasionally the velocity readings calculated from the electromagnetic flow meter may be too high for good comparative results (Table)

Acknowledgements

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SUMMARY

High speed cinefluorography of cervicocerebral blood flow patterns has been performed in 44 dogs. Under normal physiologic conditions the dog's vertebral artery has a 30 to 60% slower movement of Ethiodol droplet than the common carotid artery. Back flow in the vertebral artery was always seen while in the common carotid artery of the normotensive dog it did not occur under our experimental conditions.

ZUSAMMENFASSUNG

Bei 44 Hunden wurde Ultrarapidfilmung der Cervicocerebralen Blutzirkulation durchgeführt. Unter normalen Bedingungen ist die Fortbewegung von Ethiodoltropfen in der Arteria vertebralis des Hundes 30–60% langsamer als in der Arteria carotis communis. Ein Rückfluss war immer zu sehen in der Vertebralarterie während ein solcher in der Arteria carotis des Hundes in normalen Druckverhältnissen bei unseren Versuchsbedingungen nicht auftrat.

RÉSUMÉ

Les auteurs ont fait sur 44 chiens une cinéradiographie à grande vitesse des vaisseaux cervico-cérébraux. Dans des conditions physiologiques normales le mouvement des gouttelettes d'Ethiodol est de 30 à 60% plus lent dans l'artère vertébrale du chien que dans la carotide primitive. Ils ont toujours constaté un reflux dans l'artère vertébrale alors que dans les conditions d'expérience il ne se produit jamais dans la carotide primitive chez les chiens normotendus.

common carotid and the left vertebral artery were examined separately (in one half, the carotid was examined first and in the other, the vertebral). Then in the last 30 studies, they were examined simultaneously. In all of these examinations the droplet velocity was higher in the carotid than in the vertebral artery. To our knowledge this is the first comparative velocity study performed on these two vessels.

Since we have observed a change in droplet velocity after repeated injection of radiopaque oil, only the first few droplets have been used for plotting, to exclude the possibility of thrombosis of carotid or vertebral branches and changes in blood flow possibly thus induced. With repeated injection of oil droplet the blood velocity and consequently the blood flow decreases. The velocities obtained in three successive studies in the same dog are good examples (Figs 5, 6, 9 and 10). With the initial injection of oil droplets, the maximum common carotid droplet velocity was 90 cm/sec and that of the vertebral was 32 cm/sec (Figs 5 and 6). After injection of 1.1 ml of Ethiodol the maximum carotid velocity decreased to 73 cm/sec and that of the vertebral artery decreased to 20.0 cm/sec. After injection of 2.3 ml of Ethiodol the maximum common carotid droplet velocity further decreased to 52 cm/sec (Table).

On the basis of our hydrodynamic model experiments (Fig. 1), we make the assumption that in the range of internal vessel diameter, with which we are working, the droplet velocity is representative of the average arterial blood velocity. Moreover, the comparative studies of droplet technique and electromagnetic flow meter measurements demonstrate the validity of this assumption (Table).

Presence or absence of back flow in the carotid artery has been discussed by many authors (INOUE & KOSAKA 1959, KATZ & KOLIN 1938, MACHELLA 1936, McDONALD 1960, SHIPLEY et al. 1943). The normal presence of back flow in the carotid of anesthetized dogs has been emphasized by some authors and denied by MACHELLA and also by KATZ & KOLIN. SHIPLEY and collaborators have frequently observed back flow in the common carotid artery of dogs. INOUE & KOSAKA have only rarely found a post systolic back flow in the carotid artery of dogs. On the basis of our experiments, which produce no trauma to the artery, back flow has not been observed in normal animals in the common carotid artery. However, back flow has been observed when the dog is in an abnormal state due either to hypotension or production of thrombosis by oil injection.

The maximum flow values in the Table correspond to P_1 and the minimum flow values to V_1 as has been described by INOUE & KOSAKA.

It is noteworthy that the highest maximum flow in the common carotid artery of dogs, obtained by the electromagnetic flow meter in our experiments

THERMISTORS FOR MEASUREMENT OF CEREBRAL BLOOD FLOW

by

JAMES C BRILL AMOS NORMAN WILLIAM WEIDNER and WILLIAM HANAFEE

Thermistors are semiconductors which have a property of changing their resistance to the flow of electrical current inversely proportional to their temperature. That is to say the colder a thermistor, the greater its resistance. This property can be utilized for the measurement of blood flow using the hot wire anemometer principle (BRILL HANAFEE & NORMAN 1964 DELAUNOIS 1961)

Principle Thermistors were originally manufactured to measure temperature. After appropriate calibrations have been made the thermistor is placed in an environment of unknown temperature. Its resistance to a known electrical current can then be measured, this resistance being dependent on the temperature of the new environment.

In patients, body temperature can be accurately measured to within 0.02 to 0.03 °C. If the thermistor is inserted into the blood stream and an additional electrical current is made to flow through the instrument, the thermistor, being a resistor, will become hotter than the blood. Therefore, the colder blood flowing past the heated thermistor will take heat away from it or cool it, changing its resistance.

REFERENCES

- BONAKDARIPOUR A Principles and medical applications of electronic x ray image intensifiers Thesis Temple University School of Medicine Philadelphia (1958) 63
- COLTMAN J W Fluoroscopic image brightening by electronic means Radiology 51 (1948) 359
- CURTIS I B Rapid serial angiography Preliminary report J Neurol Neurosurg Psychiat 12 (1949) 167
- GIMENEZ J L CARTER B L STEWART G H and LYNCH P R Flow pattern analysis of particulate contrast material in blood vessels Acta radiol 50 (1961) 350
- HARDESTY W H ROBERTS B TOOF J I and ROYSTER H P Studies of carotid artery blood flow in man New Engl J Med 263 (1960) 944
- INOUE A and KOSAKA H A study of flow patterns in carotid and femoral arteries of rabbits and dogs with the electromagnetic flow meter J Physiol 147 (1959) 209
- KATZ J N and KOLIN A The flow of blood in the carotid artery of the dog under various circumstances as determined with the electromagnetic flow meter Amer J Physiol 111 (1938) 788
- LYNCH P R CARTER B I GIMENEZ J and KRINCH R Venous Carotid flow pattern in cats as studied with high speed cinefluorographic technique Amer J Physiol 199 (1960) 1139
- MACHIELIA T T The velocity of blood flow in arteries in animals Amer J Physiol 115 (1936) 632
- Mc DONALD D A Blood flow in the arteries p 133 The Williams and Wilkins Company Baltimore 1960
- MOUZ E Lencephalographie arterielle son importance dans la localisation des tumeurs cerebrales Rev neurol 2 (1927) 72
- OHILSSON N M Left heart and aortic blood flow in the dog Acta radiol Suppl 213 1961
- PETERSEN K O Personal Communication Odense Denmark
- SEARS F W and ZEMANSKY M W University Physics Third Edition Vol 1 p 67 and p 339 Addison Wesley Publishing Co Inc Reading Mass 1963
- SIMPLEY R E GRECC D E and SCHROEDER T T An experimental study of flow patterns in various peripheral arteries Amer J Physiol 138 (1943) 718
- STAUFFER H M OPPENHEIMER M J SOLOFF L A and STEWART G H Cardiac physiology revealed by the roentgen ray Amer J Roentgenol 78 (1957) 193

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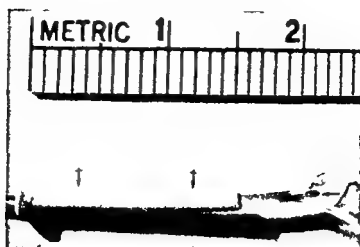


Fig 1 The three flanges of the distal end of the teflon catheter are molded to a flare by heating the teflon to 600° C for six seconds. The thermistor 0.33 mm in diameter is then wired and mounted in the distal end of the catheter (→). A small length of P90 polyethylene (→) is placed over the teflon to facilitate insertion of the flow meter into the carotid catheter. The polyethylene will cause the flanges of the flow meter to fold together.

By measuring either the resistance of the thermistor or the amount of current necessary to keep it at a higher temperature than the blood, the linear velocity of the blood stream can be measured. By accompanying these measurements with angiography, the diameter of the blood vessel, and hence its cross sectional area, can be measured from the roentgen films.

Materials The actual construction of the flow meter is simple. The 0.013 (Veco 34 A1) glass coated thermistor bead is mounted in a 100 cm long teflon tube 0.11 mm in diameter (Fig 1). The end of the teflon tube is split and splayed to center the thermistor in the vessel. The leads of the thermistor consist of 0.001 platinum wire. They are soldered to a No. 40 copper wire which runs throughout the length of the teflon catheter. The solder joints are insulated with belden wire enamel and the thermistor is bonded to the previously etched teflon tip with a special epoxy. The mechanical properties of this epoxy allow flexion of the tip without loss of the thermistor or its connections.

Electrically, the thermistor constitutes one arm of a Wheatstone bridge. When the voltage across the bridge is very low, i.e. approximately 0.1 volt, the resistance of the thermistor is determined chiefly by ambient temperature. But when the bridge voltage is increased, the thermistor begins to be heated by the extra current going through it. As it is heated, its resistance drops. The bridge is then balanced with the thermistors heated. Anything that tends to

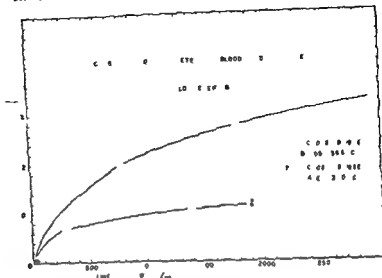


Fig. 2. R is the resistance of the thermistor in a liquid that is not flowing. ΔR is the change in resistance that occurs due to the cooling effect of either blood or water flowing past the heated thermistor. The shift of the calibration curve is apparently caused by the greater viscosity of blood.

cool the thermistor, raises its resistance thus unbalancing the bridge. The amount that the bridge is unbalanced is a measure of the linear velocity of flow to which the thermistor is exposed. Calibration of the flow meter was done both *in vitro* in water and blood and *in vivo* in the dog. The indifferent arms of the bridge are 500 Ω each. The galvanometer is a Hewlett Packard 425A microvolt meterammeter. The thermistor is about 3500 Ω at 25 $^{\circ}\text{C}$ and the variable arm of the bridge is a Leeds and Northrup 4754 Decade resistance box.

Calibration. The thermistor is placed in a still water bath at 37 $^{\circ}\text{C}$. Seven point seven volts (6.1 volt battery plus 1.6 volt battery) are placed across the bridge. The variable arm of the bridge is adjusted so that the bridge is balanced. Then various flows are circulated by the thermistor. Each of these flows will cause the bridge to unbalance. The bridge is rebalanced at that particular flow of interest. The change in resistance i.e. R_{flow} minus R_{still} is a measure of linear flow. For the convenience of using a dimensionless variable we have plotted $\frac{R_{\text{flow}} - R_{\text{still}}}{R_{\text{still}}}$. We have done the same sort of calibration circulating blood of 37 $^{\circ}\text{C}$ past the flow meter (Fig. 2).

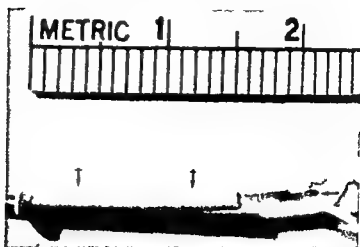


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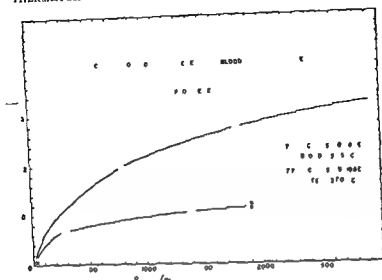


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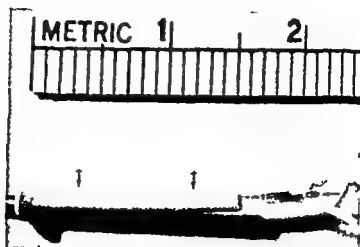


Fig 1 The three flanges of the distal end of the teflon catheter are molded to a flare by heating the teflon to 600 C for six seconds. The thermistor 0.33 mm in diameter is then wired and mounted in the distal end of the catheter (\rightarrow). A small length of P90 polyethylene (\leftrightarrow) is placed over the teflon to facilitate insertion of the flow meter into the carotid catheter. The polyethylene will cause the flanges of the flow meter to fold together.

By measuring either the resistance of the thermistor or the amount of current necessary to keep it at a higher temperature than the blood, the linear velocity of the blood stream can be measured. By accompanying these measurements with angiography, the diameter of the blood vessel, and hence its cross sectional area, can be measured from the roentgen films.

Materials The actual construction of the flow meter is simple. The 0.013 (Veco 34 A1) glass coated thermistor bead is mounted in a 100 cm long teflon tube 0.8 mm in diameter (Fig 1). The end of the teflon tube is split and splayed to center the thermistor in the vessel. The leads of the thermistor consist of 0.001 platinum wire. They are soldered to a No. 40 copper wire which runs throughout the length of the teflon catheter. The solder joints are insulated with belden wire enamel and the thermistor is bonded to the previously etched teflon tip with a special epoxy. The mechanical properties of this epoxy allow flexion of the tip without loss of the thermistor or its connections.

Electrically, the thermistor constitutes one arm of a Wheatstone bridge. When the voltage across the bridge is very low, i.e. approximately 0.1 volt, the resistance of the thermistor is determined chiefly by ambient temperature. But when the bridge voltage is increased, the thermistor begins to be heated by the extra current going through it. As it is heated its resistance drops. The bridge is then balanced with the thermistors heated. Anything that tends to

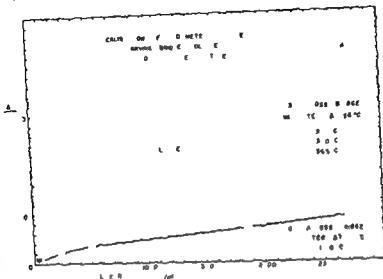


Fig. 4. Calibrations were made at varying temperature of the water bath to see if variation in the patient's body temperature would interfere with flow measurements. In general the differences are less than 5 per cent. Similar observations were made in blood.

measurement takes approximately four to five minutes. A film is exposed of the vessel filled with contrast material. The cross sectional area of the vessel is computed by first calculating the magnification factor from radiographed diameter of the catheter versus its actual measured diameter. The internal diameter of the artery in question is measured by contrast material and then reduced by the magnification factor. From our calibration we constructed a graph of $\frac{R_i - R_o}{R}$ versus linear velocity. The measurement taken in the carotid artery is simply referred to the graph and the linear velocity is read off the graph thus:

Linear velocity \times cross sectional area of carotid artery = volume flow in the artery

Pitfalls

There are some anticipated questions to the use of this method

1. *Centering* In water no difference could be detected whether the device is centered or not with linear velocities up to 2.500 cm/min. With blood in vivo centering does make a difference of 120 cm/min near the wall of the vessel.

2. *Direction of flow* If the flow meter is heading into the water stream it

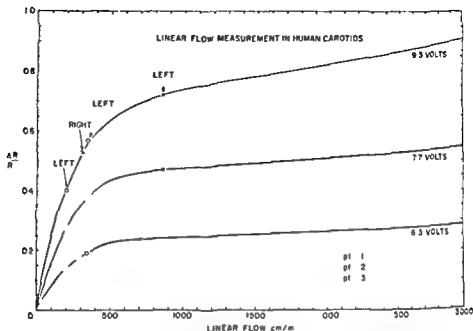


Fig 3 Three separate measurements are made in each artery with varying voltages across the thermistor. The voltage changes cause a greater differential in temperature between blood and thermistor. By measuring the cross sectional area of the internal carotid arteries in the neck, the volume flow can be calculated by cross sectional area \times linear flow. The results were as follows:
 Patient I Left internal carotid artery 80 cc/min
 Patient II Left internal carotid artery 18 cc/min
 Patient III Right internal carotid artery 96 cc/min Left internal carotid artery 230 cc/min

Blood is more viscous than water. Convection currents in blood are slower than in water. For the same voltage across the bridge, the thermistor will be heated to a higher temperature in blood than in water. Thus, there are significant calibration differences between water and blood. The flow meter is more sensitive for the same bridge voltage in blood than in water. Sensitivity of the flow meter is determined by the difference in temperature of the thermistor and the flowing stream. Obviously, the more voltage across the bridge, the hotter will be the thermistor, and thus the flow meter will be more sensitive.

In our system we have taken $\frac{R_f - R_o}{R}$ as a measure of sensitivity.

In practice, flow measurement is achieved by slipping the teflon catheter inside an angiographic catheter previously located in the carotid artery by percutaneous technique (HANAFEE & WEIDNER 1963). The teflon catheter is fed through the angio catheter until its tip emerges in the blood stream. The bridge voltage is applied and the bridge is balanced (Fig 3). The balance resistance is noted and the thermistor is withdrawn. The whole process of

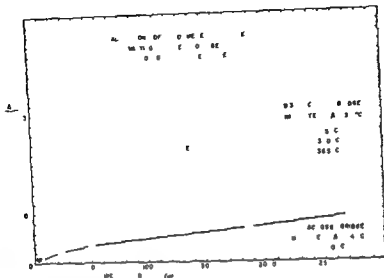


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Linear velocity \times cross sectional area of carotid artery = volume flow in the artery

Pitfalls

There are some anticipated questions to the use of this method.

1 *Centering* In water no difference could be detected whether the device is centered or not with linear velocities up to 2500 cm/min. With blood in vivo centering does make a difference of 120 cm/min near the wall of the vessel.

2 *Direction of flow* If the flow meter is heading into the water stream it

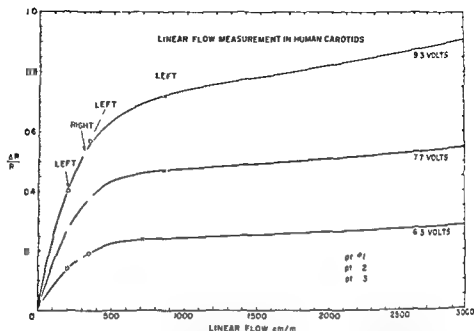


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- Patient I Left internal carotid artery 80 cc/min
 □ Patient II Left internal carotid artery 48 cc/min
 x Patient III Right internal carotid artery 96 cc/min Left internal carotid artery 230 cc/min

Blood is more viscous than water. Convection currents in blood are slower than in water. For the same voltage across the bridge, the thermistor will be heated to a higher temperature in blood than in water. Thus, there are significant calibration differences between water and blood. The flow meter is more sensitive for the same bridge voltage in blood than in water. Sensitivity of the flow meter is determined by the difference in temperature of the thermistor and the flowing stream. Obviously, the more voltage across the bridge, the hotter will be the thermistor, and thus the flow meter will be more sensitive. In our system we have taken $\frac{R_t - R_0}{R_0}$ as a measure of sensitivity.

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will record 5 % more linear velocity at 2 500 cm/min than it will if the stream is coming from behind

3 *Stability and drift* If thermistors are used only for temperature measurement, drift is guaranteed by the manufacturer at less than 0.1 per cent. However, by running a current of 8 to 10 milliamperes through the thermistor to produce milliamperes through the thermistor to produce self heating, several changes seem to occur. Its resistance varies from day to day at specific temperatures. In addition, calibration curves tend to change in configuration. One explanation of these changes may be based on sluffing of oxygen molecules bound to the metallic oxides of thermistor beads.

4 *Thermistor care* Care must be taken not to overheat the thermistor, as this will change its physical properties. Sudden surges of current cause an "arcing effect" which may melt the small electrical leads.

5 *Clotting and coagulation of the blood* The thermistor has been heated 30°C above the temperature of still oxygenated blood without causing coagulation. Although the thermistor is hot, the actual heat transfer is very small. Clotting dangers may be minimized by (1) siliconizing the catheter tip, and (2) avoiding prolonged measurement.

6 *Change in body temperature* According to our data, we can tolerate a shift in the temperature of the bath or of the blood of as much as 1.0°C without any detectable effect. Our calibration is accurate from 36.5°C to 37.5°C (Fig. 4). Such gross fluctuations are not likely to occur during the short measuring interval.

7 *Arterial spasm and turbulent flow* Localized spasm or stenosis can cause marked variations in velocity throughout the length of the carotid artery. Since the thermistors are not radiopaque their precise location cannot be determined.

Conclusions

Although thermistors offer a new technique for non operative measurement of blood flow, several handicaps prevent their widespread use. The drift and lack of stability of thermistors means that frequent calibrations are essential. In our hands, one week is the longest period of time one calibration has remained in use. This also varies considerably between individual thermistor beads. Calibrations must be made in blood and an average time of five hours has been required for completing our curves.

We hope to overcome some of these problems in the near future by use of higher inherent resistance thermistor beads to yield greater stability. In addition improved electronics may change thermistors from a valuable research tool to a daily clinical measurement.

Acknowledgement

This paper was written with the technical assistance of Mr Jon M Shinn

SUMMARY

Thermistors offer an additional means of measuring volume blood flow. Our instrument can conveniently be inserted at the time of carotid angiography if the catheter technique is used.

ZUSAMMENFASSUNG

Thermistoren sind ein Hilfsmittel zur Messung des Blutvolumens. Unser Instrument kann bei Anwendung der Kathetertechnik mit Vorteil während der Carotis-Angiographie angebracht werden.

RÉSUMÉ

Les thermistors offrent un nouveau moyen de mesurer le débit sanguin. Si on utilise le cathétérisme, notre instrument peut être introduit au cours de l'angiographie carotidienne.

REFERENCES

- BRILL J, HANAFEE W and NORMAN A. A flowmeter for use in angiographic studies. *Radiology* 87 (1964) 133.
DELAUNOIS A L. Continuous measurement of blood flow and cardiac output by means of a Courmand catheter equipped with thermistors. *Arch int Pharmacodyn* 134 (1961) 245.
HANAFEE W and WEIDNER W. Selective carotid angiography. *Radiology* 81 (1963) 589.

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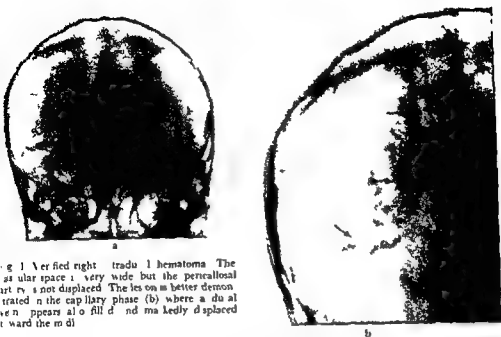


Fig. 1. Verified right parietal extradural hematoma. The avascular space is very wide but the pericallosal artery is not displaced. The lesion is better demonstrated in the capillary phase (b) where a dual venous phase also fills and markedly displaces it toward the midline.

We now wish to emphasize another diagnostic element—the discrepancy between the width of the avascular space and the displacement of the midline blood vessels, i.e., the pericallosal artery and internal cerebral vein.

This radiographic sign was observed by CROWQVIST & KOHLER (1963) in 9 out of 25 extradural hematomas (3 temporal lateral, 5 parietal, and 1 occipital). In other cases, such as in 5 temporal basal hematomas, the avascular space was not clearly visible, but the displacement of the pericallosal artery and of the internal cerebral vein was less than that of the Sylvian vessels. The avascular space was absent in 3 out of 4 frontal hematomas, while in the case of an occipital effusion it was possible to demonstrate it only by means of an oblique projection. Of the 25 patients, 7 had multiple intracranial traumatic lesions; it is not always clear which data refer to the simple rather than to the complicated hematomas.

Material. Our material consists of 21 acute traumatic extradural hematomas which were verified at surgery; of these, 16 were simple and 5 were associated with other lesions: two with cerebral laceration, two with cerebral laceration and subdural hematoma, and one with subdural hematoma. All cases were diagnosed preoperatively as extracerebral expanding processes. Among the

ANGIOGRAPHIC TECHNIQUE AND DIAGNOSIS IN BRAIN LACERATIONS AND EXTRADURAL HEMATOMAS

by

PIETRO DETTORI and GIOVANNI RUGGIERO

For a number of years our group has devoted particular attention to neurotraumatology and to radiological diagnosis of acute head injuries (RUGGIERO 1962). The present study deals with two problems: 1) the correct diagnosis of extracerebral hematomas, and 2) a particular angiographic technique in cases of acute head injuries, which has proved of special interest in brain lacerations.

The most important criteria in the differential diagnosis of extracerebral effusions were presented in a preliminary note at the Tenth International Congress of Radiology in Montreal in 1962 (RUGGIERO et coll. 1964). They are summarized below:

	Subdural hematomas	Extradural hematomas
Frontal view	Hemispherical outlining of the vascular space smooth and regular Absence of displacement of branches of the middle meningeal artery	Hemispherical outlining of the vascular space irregular and indented Displacement toward the midline of the branches of the middle meningeal artery
Lateral view	Normal	Signs of space occupying lesion

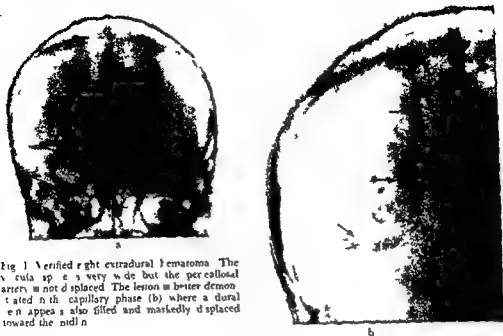


Fig 1 Verified right extradural hematoma. The avascular space is very wide but the pericallosal artery is not displaced. The lesion is better demonstrated in the capillary phase (a) where a dural vein appears also filled and markedly displaced toward the midline.

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Material. Our material consists of 21 acute traumatic extradural hematomas which were verified at surgery: of these, 16 were simple and 5 were associated with other lesions, two with cerebral laceration, two with cerebral laceration and subdural hematoma, and one with subdural hematoma. All cases were diagnosed preoperatively as extracerebral expanding processes. Among the

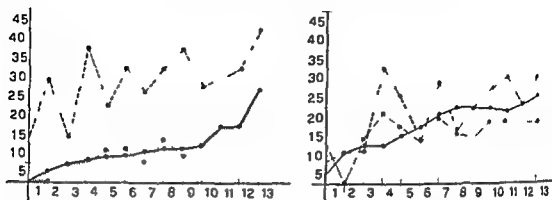


Fig 2 Ratio between width of vascular space (interrupted line) and displacement of the pericallosal artery (solid line) and internal cerebral vein (dotted line) in mm. Subdural hematomas on right; extradural on left

simple extradural hematomas 13 were diagnosed as such, while in the remaining 3 an unsatisfactory technique did not allow a good evaluation of the radiograms

Discussion

In our series all *simple extracerebral hematomas* showed a displacement of the pericallosal artery and the internal cerebral vein which was always less than the width of the vascular space (Fig 1). For a better evaluation of this sign, data from 13 extradural hematomas have been compared with those from a similar number of subdural lesions (Fig 2).

In cases of extracerebral hematomas complicated by other lesions such as contusions or lacerations the relationship is necessarily altered. It is obvious that an intracerebral lesion which is expanding will tend to reduce the vascular space due to an associated extracerebral hematoma, while it will increase the displacement of the midline vessels toward the opposite side, for this reason the discrepancy between the width of the vascular space is of diagnostic value only in those extracerebral hematomas which are not associated with other lesions.

When we are confronted with *cerebral lacerations*, especially if small, comparison between right and left angiographies may be necessary (Fig 3). There are two possibilities: 1) puncture of both carotids prior to the taking of any radiograms, this will allow bilateral angiographies in the same projection, but arterial puncture of the contralateral carotid may prove to have been unnecessary; 2) after the first angiography, puncture of the contralateral carotid without moving the head, thus performing the second angiography under identical radiographic conditions. This is the technique we have

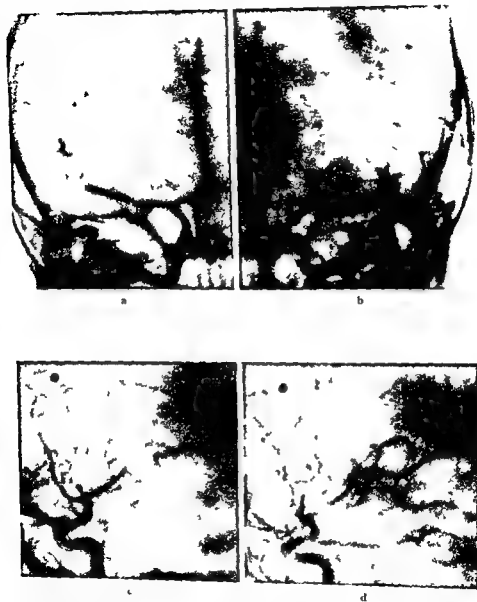


Fig 3 Small right to left lateral view. The smallest difference in the aspect of the right (a) and left (b) middle cerebral arteries and the right (c) and left (d) carotid siphons can be appreciated since the projections are identical.

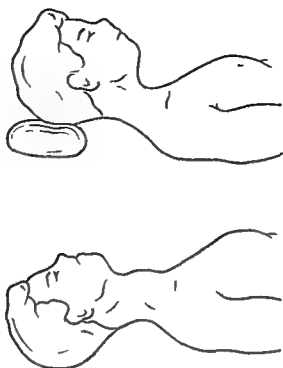


Fig. 1. Ideal position for puncturing the internal or common carotid artery (bottom). Position suitable in cases of cranial trauma (above).

adopted, in spite of its difficulties. If the patient is uncooperative, general anesthesia is necessary, one must learn to puncture the artery with the patient's head slightly flexed (Fig. 4), this being far from the hyperextended position. For this reason we always try to puncture carotids without extending the patient's head, even in non traumatic cases. The flexed head technique is also useful in patients with intracranial tumors in whom hyperextension of the head, by raising venous tension, can increase intracranial pressure.

SUMMARY

The ratio between the displacement of the pericardial artery and internal cerebral vein and the width of the vascular space in extracerebral hematomas is analyzed. The value of this sign in differentiating extra and subdural hematomas is emphasized. The technique of carotid puncture in head injuries is discussed. A position with the head flexed being recommended.

ZUSAMMENFASSUNG

Bei extracerebralen Hamatomen wird das Verhältnis zwischen der Dislokation der Art pericallosa und Vena cerebralis und der Weite des avaskularen Spatiums analysiert. Der Wert dieses Zeichens für die Differenzierung von extra und subduralen Hamatomen wird hervorgehoben. Die Technik der Carotidpunktion bei Schädelverletzungen wird besprochen, wobei eine Flexionsstellung des Schädels empfohlen wird.

RÉSUMÉ

Dans le but de préciser le diagnostic des hématomes extraduraux, les auteurs analysent le rapport entre ampleur de l'espace avasculaire et déplacement de l'artère pericallosale et veine cérébrale interne. En discutant la technique angiographique, les auteurs proposent dans les cas de traumatisme crânien de ponctionner l'artère carotide interne ou primitive dans une position tête fléchie.

REFERENCES

- CRONQVIST S and KOHLER R. Angiography in epidural haematomas. *Acta radiol* 1 (1963) 47.
- RICCIARDO G. Il quadro radiologico del cranio acuto. Rapporto al Congresso della Società dei Neurologi Ospedalieri Roma 1967.
- CALABRO A, METZGER J and SIMON J. Arteriography of the external carotid artery. *Acta radiol* 1 (1963) 395.
- LEIGHTON R S, DETTORI P and COLLINELLA F. Acute cranial trauma. *Acta radiol* 2 (1964) 487.

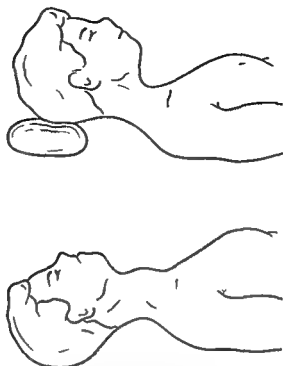


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ZUSAMMENFASSUNG

Bei extracerebralen Hämorrhagien wird das Verhältnis zwischen der Dura mater, der Arteria ptericallosa und Vena cerebri interna mit der Werte des avaskulären Spatiums analysiert. Der Wert dieses Zeichens für die Differenzierung von extra- und subduralen Hämorrhagien wird hervorgehoben. Die Technik der Carotisangiographie bei Schädelverletzungen wird beschrieben, wobei eine Flexionsstellung des Schädels empfohlen wird.

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Dans le but de préciser le diagnostic des hémorragies extracrâniennes, les auteurs analysent le rapport entre l'ampleur de l'espace avasculaire et déplacement de l'arterio-venose ptericallosa et de la veine cérébrale interne. En discutant la technique angiographique les auteurs proposent dans les cas de traumatisme crânien de ponctionner l'artere carotide interne ou externe dans une position tête fléchie.

REFERENCES

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LA SOUSTRACTION APPLIQUEE A L'ETUDE ANGIOGRAPHIQUE DES LESIONS DE LA FOSSE POSTERIEURE

par

D. DILENCI et J. MITZNER

L'art de soustraction que nous utilisons comme complément de l'angiographie depuis 1961 est devenu ces derniers temps une technique de routine.

Les artériogrammes en sont particulièrement nets pour l'angiographie vertébrale et ceci non seulement pour l'identification des vaisseaux veineux et artériels, mais aussi dans l'appréciation de la vascularisation des lésions néoformées de la fosse postérieure.

L'étude de 32 lésions de la fosse postérieure prouve que grâce à une soustraction correcte, les vaisseaux sans la superposition osseuse particulièrement gênante au niveau des rochers (tant en projection de face que de profil) se laissent reconnaître avec netteté sur tout leur trajet, les vaisseaux néoformés peuvent ainsi se détacher nettement du fond osseux atténué.

Parmi ces 32 lésions, on trouve 11 neurinomes de l'acoustique, 5 méningiomes (3 de l'angle, 1 du clivus et 1 de la convexité cérébelleuse), 3 angioréticulomes, 7 angiomes ou anévrysmes du tronc basilaire, 2 cas de traumatisme.

Pour chaque type de lésion nous avons cherché à dégager les détails angio-



Fig 1 Angiome cérébelleux La projection antéro-postérieure retrouve grâce à la soustraction tout son intérêt même en angiographie vertébrale elle dégage en plus des détails peu visibles sur les clichés conventionnels d'autres mal rendus par la projection de profil Dans ce cas il est possible par exemple d'individualiser sur l'angiogramme de profil (a) tous les noyaux opaciques objectifs ou sur l'angiogramme de face (b)

graphiques que la soustraction a permis d'obtenir en plus par rapport à l'angiographie conventionnelle

Soulignons tout d'abord l'intérêt que nous avons porté à la projection de face dans les cas où il était possible de pratiquer la soustraction pour les deux projections Les images d'opacification pathologique dans plusieurs de ces cas étaient peu visibles sur l'angiogramme de profil mais nous les avons retrouvées parfois très nettement sur l'angiogramme de face (Fig 1)

La raison en est que sur l'angiogramme de profil, la superposition des deux lobes cérébelleux au temps capillaire produit une opacité du parenchyme dans laquelle l'image pathologique peut se perdre complètement L'opacité cérébelleuse ainsi devient significative dans la fosse postérieure et il est facile d'en apprécier l'importance dans les cas où l'ombre cérébelleuse apparaît que d'un côté seulement

Nous n'avons trouvé aucune opacification particulière dans les 11 cas de neurinome de l'acoustique Sur les angiogrammes de face dans 3 d'entre eux cependant nous avons pu noter un vaisseau périluminal avec une disposition en vaisseaux bordants des branches artérielles peritumorales (Fig 2)

Dans un seul cas la tumeur dont les limites paraissaient nettement définies par un de ces vaisseaux bordants apparaissait moins transparente que les tissus avoisinants

Dans les cas de méningiome la soustraction a permis de vérifier le manque

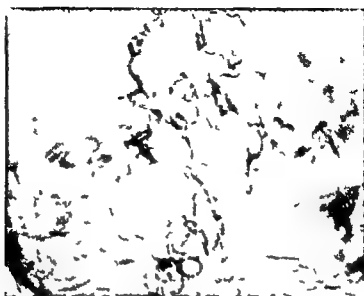


Fig. 2. Neurinome de l'acoustique à droite. Les vaisseaux voisins sont disposés en vaisseaux bordants.

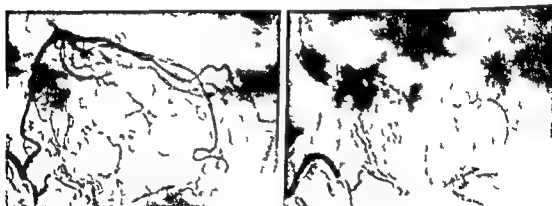


Fig. 3. Gros méningiome de la convexité du cervelet. La branche méningée de l'artère vertébrale est particulièrement développée et à ses dépens semblent s'être formés des néo-vaisseaux.

de vascularisation pathologique par l'artère vertébrale lorsque la tumeur est située dans la partie antérieure de la fosse postérieure.

Dans le seul cas où la tumeur est située dans la partie postérieure de la fosse postérieure, nous avons constaté une vascularisation pathologique aux dépens d'une branche méningée postérieure (Fig. 3).

Parmi les tumeurs intracérébelleuses, nous avons pu mettre en évidence des vaisseaux néo formés dans un médulloblastome et dans une métastase. Dans le médulloblastome, on nota tout d'abord une hypertrophie de l'artère cérébelleuse moyenne gauche et ensuite des vaisseaux néo formés. L'aspect

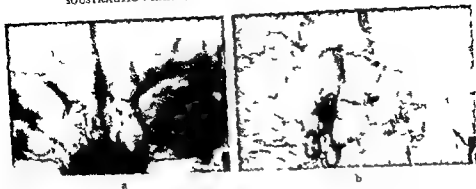


Fig 4 a) Aucune image certaine de vascularisation pathologique n'est mise en évidence sur l'angiogramme conventionnel b) La soustraction nous permet de noter la présence de vaisseaux faisant partie d'une hypervascularisation pathologique au niveau de la lésion



Fig 5 Ces deux clichés qui ont été obtenus avec une projection qu'on ne peut utiliser qu'avec la technique objective bien des contours latéraux et postéro inférieurs des lobes cérébelleux

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L'étude des tumeurs et des malformations vasculaires est particulièrement favorisée par la soustraction puisque nous avons pu dégager facilement les pédicules afférents. La soustraction a été particulièrement utile dans les cas où ces pédicules ne s'identifiaient pas avec une artère cérébelleuse et où ils étaient en partie masqués par la superposition de l'os occipital dans un cas d'angiome de la tente le pédicule était constituée par une branche de la meningée postérieure

Dans un cas d'angiome cérébelleux le malade fut opéré avec le diagnostic de tumeur cérébelleuse à la suite d'un examen pneumographique et todo-

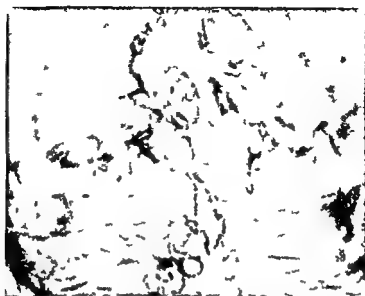


Fig. 2. Neurinome de l'acoustique à droite. Les vaisseaux voisins sont disposés en vaisseaux bordants.

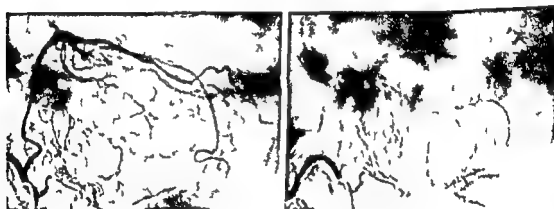


Fig. 3. Gros méningiome de la convexité du cervelet. La branche méningée de l'artère vertébrale est particulièrement développée et à ses dépens semblent se former des néo vaisseaux.

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Fig 4 a) Vue en image certaine de vascularisation pathologique mise en évidence sur l'angio-gramme conventionnel b) La soustraction nous permet de noter la présence de vaisseaux faisant preuve d'une hyper-vascularisation pathologique au niveau de la lésion



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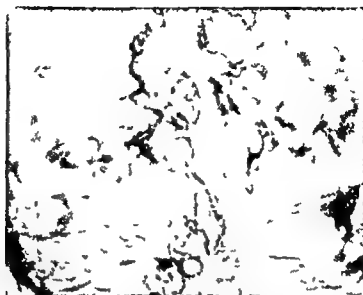


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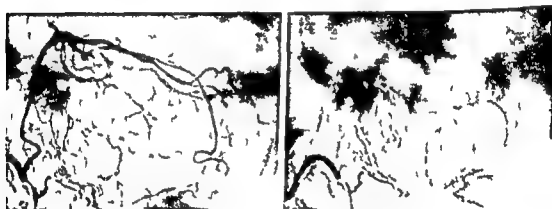


Fig 3 Gros méningiome de la convexité du cervelet. La branche méningée de l'artère vertébrale est particulièrement développée et ses dépens semblent s'être formés des néo vaisseaux

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En objectivant le shunt artério veineux l'angiographie et elle seule a permis une approche satisfaisante des angiomes cérébraux et parallèlement, l'élaboration d'une tactique chirurgicale dont le succès prouve l'efficacité.

En matière de malformations vasculaires médullaires la période anatomoclinique se prolonge encore. La nomenclature proposée par WILBURN MASSON (36) en 1943 demeure la plus couramment utilisée dans les publications de ces

ventriculographique. L'angiographie vertébrale qui fut malheureusement pratiquée du côté opposé, nous émit une image négative sur les clichés conventionnels, sans aucune neoformation vasculaire particulière. La soustraction pratiquée après l'intervention a permis de découvrir malgré tout, plusieurs vaisseaux de la neoformation (Fig. 4).

Dans les deux cas de traumatisme, la symptomatologie était en faveur d'une lésion de la fosse postérieure et l'angiographie avec et sans soustraction fut négative. Dans l'un de ces cas qui fut opéré on trouva un hématome intra-cérébelleux.

À ce propos, nous désirons souligner, que grâce à la soustraction il devient possible de découvrir d'éventuels décollements corticaux. Le cerveau, opacifié lui-même et bordé par les veines, repoussé par l'hématome, doit laisser libre de tout vaisseau l'espace de décollement (Fig. 5).

RÉSUMÉ

Les auteurs ont appliqué la soustraction à l'angiographie vertébrale de face et de profil dans 32 lésions de la fosse postérieure. Ils soulignent l'intérêt de cette technique pour l'étude de la vascularisation des méningiomes, des tumeurs malignes et des malformations vasculaires et pour le diagnostic des hématomes extra-cérébelleux.

SUMMARY

The subtraction method was used for vertebral angiography for both antero-posterior and lateral films in 32 lesions of the posterior fossa. The value of the technique is stressed for the study of the vessels in meningiomas, malignant tumours and vascular malformations as well as for the diagnosis of extra-cerebellar hematomas.

ZUSAMMENFASSUNG

Die Subtraktion wurde in 32 expansiven Prozessen der hinteren Schädelgrube in frontalen und lateralen Vertebral-Angiogrammen angewendet. Es wird auf den Wert der Methode für die Beurteilung der Blutversorgung der Meningiomen, der bösartigen Tumoren und der Gefäßmissbildungen und für die Diagnose der extracerebellären Hämatomen hingewiesen.

BIBLIOGRAPHIE

- DAVID M., DILENGE D. et BRIHAYE J. Les progrès réalisés dans la connaissance des malformations artério-veineuses cérébrales. Importance des techniques angiographiques. *Acta chir. belg.* 9 (1963) 961.
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Fig. 7. Cas 2. Aortographie par voie fémorale. Face centrée sur le cou. Soustraction. L'angiome se projette sur C4—C5. Pedicules multiples issus des vertébrales droite et gauche et des branches du tronc thyro-hi-scapulaire des deux côtés.

de la méthode de soustraction de ZIEDZES DES PLANTES (37) ce procédé photographique améliore la qualité des images et en facilite la lecture.

Les observations suivantes illustrent les possibilités de ces explorations angiographiques.

Observations

Cas 1. Femme de 22 ans. Début clinique à 12 ans. Actuellement triplégie spasmodique avec troubles urinaires. Les clichés simples du rachis et la myélographie lipidolée font porter le diagnostic d'angiome médullaire en C4—C6. Une phlébographie rachidienne est négative.

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BIBLIOGRAPHIE

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En matière de malformations vasculaires médullaires la période anatomique se prolonge encore. La nomenclature proposée par WYBURN MASON (36) en 1943 demeure la plus couramment utilisée dans les publications de ces

vingt dernières années. L'artérisme racémeux veineux, l'aneurysme artériel veineux, les anomalies artérielles y sont différenciés sur des critères statiques, visuels ou anatomiques. Cette hétérogénéité paraît bien paradoxale en comparaison avec l'énorme prédominance des malformations artério-veineuses décelées par l'angiographie au niveau du cerveau. C'est pourquoi bien des auteurs ont récemment mis en doute la valeur de cette classification.

Le peu d'intérêt accordé aux études angiographiques nous paraît significatif de la force des idées reçues. HENSON & CROFT (18) dans leur publication de 1956, rapportent les premiers à notre connaissance, le cas d'un anéurysme artério-veineux cérébral mis en évidence par angiographie cérébrale des mai 1953.

RAND & RAND (30) en 1960 commentent dans leur cas n° 2 l'intérêt et les risques de l'ortographie qui leur a permis d'objectiver en 1954 un angiome de la jonction dorso lombaire. Leurs réflexions ne devaient trouver que peu d'échos puisque seuls DI CHIRO (8) en 1957, HOOK & IDVALL (19) en 1958, et MORRIS (28) en 1960 utilisaient l'angiographie vertébrale pour étudier quelques cas isolés cervicaux d'angioréticulome ou de malformations vasculaires. Depuis 1961, date à laquelle nous avons étudié notre premier cas d'angiome médullaire, nous avons entrepris une étude angiographique systématique de tous les cas de malformations vasculaires médullaires qui nous ont été confiés. C'est le résultat de cette enquête que nous rapportons ici.

Nous serons volontairement brefs sur les données étiologiques et cliniques et soulignerons seulement quelques points : 1) l'atteinte prédominante du sexe masculin (8♂, 1♀), 2) la précocité des troubles, fait qui va à l'encontre de l'extrême rareté de la mise en évidence de ces malformations chez l'enfant, souvent affirmée par la littérature, 3) la constance des manifestations douloureuses qui représentent souvent le premier signe et qui demeurent longtemps isolées, 4) l'évolution par poussées plus ou moins complètement régressives, ce mode évolutif a été noté neuf fois sur douze, 5) la relative rareté des hémorragies sous arachnoïdiennes (trois cas sur douze) est conforme aux données de la littérature, contrairement à ce que l'on pourrait penser ces hémorragies sont loin de faciliter le diagnostic, bien au contraire elles orientent souvent à tort vers une malformation cérébrale et font pratiquer en premier lieu des angiographies carotidiennes et vertébrales.

Les examens radiologiques classiques apportent souvent au diagnostic des arguments d'appoint ou de certitude.

Sur les clichés simples, l'élargissement du canal rachidien, l'augmentation des distances inter-pédiculaires, l'amincissement d'un ou plusieurs pédicules traduisent la présence d'un processus expansif intrarachidien, ces modifications ont été observées essentiellement dans les localisations cervicales ou

cervico dorsales (2 cas sur 4) et dorsales (4 cas sur 4), plus rarement d'ins la region du cone terminal. Une scoliose a ete notée d'ins 2 cas.

La presence de calcifications rapportée une fois par WALKER (35), l'association classique mais rare d'un angiome vertebraal sont des arguments de presumption en faveur d'un angiome medullaire. Mentionnons aussi la dilatation de la crosse de la veine azygos que nous avons retrouvée à posteriori sur les clichés simples de deux de nos malades à la lumiere des angiographies, dans des localisations cervico dorsale et dorsale.

La myelographie aux contrastes positifs constituait jusqu'à maintenant le moyen diagnostique le plus sûr. un blocage plus ou moins net de la bille opaque revelait la lesion dont la nature angiomateuse était prouvée par la presence de moulages vasculaires. GUILLAIN & AJAJOUANINE (16) furent les premiers à decrire cette image en 1925. cependant elle n'est pas constante meme en s'aidant de precautions techniques et en utilisant un index opaque volumineux comme le conseille LOUBARDI & MICELIACCA (27) il est des cas ou la myelographie revele un blocage isole, la nature vasculaire de l'obstacle etant decouverte au moment de l'intervention. Outre ses risques, sur lesquels nous ne nous etendrons pas, la myelographie a l'inconvenient de ne pas apporter de preuve formelle. Des images de moulages vasculaires sont quelquefois observées au voisinage de certaines tumeurs intra rachidiennes avec stase veineuse. on les voit egalement dans certaines coarctations aortiques.

Ces imperfections de la myelographie lipiodolée sont mentionnées par beaucoup d'auteurs dont certains tels SVEN & BAKER (33) deplorent l'impossibilite d'utiliser pour la moelle les procedes angiographiques si fructueux pour l'etude de l'encephale. Ces auteurs rapportent les echecs de leurs tentatives de phlebographie intra rachidienne fautes dans le but d'opacifier ces malformations. Precisons que pour notre part dans les trois cas ou nous avons utilise la phlebographie nous n'avons pas pu injecter l'angiome.

C'est en fonction des connaissances actuelles de la vascularisation medullaire et par assimilation avec les malformations arterio veineuses cerebrales que nous avons decide dans un cas examine par phlebographie en 1961 de faire dans le meme temps operatoire profitant de l'anesthésie generale une aortographie. Le succes de cet examen nous a incité à poursuivre nos efforts dans ce sens.

En fonction de notre experience et compte tenu des conditions anatomiques d'irrigation arterielle de la moelle, les explorations arteriographiques de cet organe doivent se faire de la maniere suivante.

1) pour le segment cervical ou cervico dorsal il faut opacifier separement les deux arteres sous clavières et la portion terminale de la crosse aortique. Du cote droit la sous claviere est examinée par ponction directe du coté



Fig 1 Cas 1 Angiographie sous claviers gauche a) Temps artériel Cliché de face centré sur l'angiome Soustraction La malformation reçoit deux afférences l'une née de la vertébrale l'autre de la cervicale ascendante b) and c) Temps artériel Clichés de face et de profil centrés sur la partie haute du cou Soustraction Les artères spinales antérieure et postérieure fournissent des afférences au pôle supérieur de la malformation

gruiche elle est étudiée soit par ponction directe, soit mieux par cathétérisme rétrograde à partir de la fémorale, la torte est opacifiée par cathétérisme rétrograde

2) pour le segment dorsal, nous faisons une aortographie thoracique, pour la région du cône terminal une aortographie thoraco-abdominale. Là encore, nous utilisons le cathétérisme fémoral rétrograde, selon le procédé de SELDINGER (32)

La qualité des renseignements fournis par ces angiographies dépend du soin apporté à la réalisation des examens. Nombre d'impératifs techniques sont à respecter, bien connus des radiologistes habitués aux examens angiographiques. Nous soulignerons seulement quelques points essentiels. L'intérêt de l'anesthésie générale, la nécessité d'une étude sérographique de durée assez longue (12 à 15 secondes), la nécessité d'explorer pour un territoire donné, tout les vaisseaux susceptibles d'apporter des afférences à la malformation, l'intérêt primordial



Fig 2 Cas 2 Angiographie par voie fémorale Face centrée sur le cou Soustraction L'angiome se projette sur C4—C5 Pédicules multiples issus des vertébrales droite et gauche et des branches du tronc thyro-bi scapulaire des deux côtés

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2) pour le segment dorsal, nous faisons une aortographie thoracique, pour la région du cône terminal une aortographie thoraco-abdominale. Là encore, nous utilisons le cathétérisme fémoral rétrograde, selon le procédé de SELDINGER (32)

La qualité des renseignements fournis par ces angiographies dépend du soin apporté à la réalisation des examens. Nombre d'impératifs techniques sont à respecter, bien connus des radiologistes habitués aux examens angiographiques. Nous soulignerons seulement quelques points essentiels: l'intérêt de l'anesthésie générale, la nécessité d'une étude sérographique de durée assez longue (12 à 15 secondes), la nécessité d'explorer pour un territoire donné tous les vaisseaux susceptibles d'apporter des artères à la malformation, l'intérêt primordial



Fig 3 Cas 6. Angiographie par voie fémorale cathéter au niveau de D6. a) Temps artériel précoce. Sous action de face. Deux afférences nourrissent cet angiome : l'une volumineuse venue de la 3^{ème} intercostale droite (→) l'autre accessoire venue de la 2^{ème} intercostale droite (→). b) Temps artériel tardif. Sous action. La masse angiomateuse est très étendue déjà opacifiée les premières efférences. c) Temps veineux. Sous action. Extrême étendue et complexité des efférences veineuses qui aboutissent aux plexus extrarachiens et à la veine azygos latérale.

supérieur de l'angiome. Du côté droit, une afférence principale née en regard de D₆ fournit en regard du 3^e espace intercostal une branche volumineuse à destination intrarachidienne qui atteint l'angiome à sa partie moyenne. Une afférence accessoire provient de la 4^e artère intercostale droite. Le shunt artério-veineux est authentifié par l'apparition précoce de veines efférentes essentiellement inférieures sur les temps tardifs. On peut suivre l'une de ces veines volumineuses dans le canal rachidien jusqu'au niveau du rachis dorsal inférieur.

Cet adolescent meurt quelques semaines plus tard à la suite d'une nouvelle hémorragie méningée. La vérification anatomique apporte plusieurs précisions intéressantes : elle confirme l'existence d'un angiome vertébral en D₄ soupçonné sur les radiographies simples du rachis ; elle découvre un angiome au niveau du foie et elle permet de pratiquer des coupes sérieuses de la moelle qui montrent l'absence d'hémorragie à l'intérieur de la moelle ; la situation essentiellement extramédullaire et postérieure des vaisseaux anormaux dont la pénétration dans la moelle se limitait à la région dorsale supérieure ; l'existence de thromboses disséminées à plusieurs endroits de la malformation.

Cas 5. Garçon de 6 ans. Apparition brutale en fin de journée de douleurs thoraciques droites puis dans la nuit de céphalées. En moins de 48 heures se constitue un syndrome de Brown Sequard. La paralysie du membre inférieur droit est totale. L'hypoesthésie gauche remonte jusqu'à D₄ ; on note un signe de Babinski bilatéral, une abolition des réflexes cutanés abdominaux et crémasteriens, une rétention d'urine.

centrée sur le crâne fut discuter un possible anévrysme du tronc basilaire mais un examen plus approfondi de la région cervicale haute montre sur ce film les artères spinales dilatées.

Myélographie — le lipiodol injecté par voie lombaire marque un temps d'arrêt en C6 puis moule des images vasculaires typiques en C5 et C4.

On fait une nouvelle angiographie vertébrale droite par ponction sous clavière centrée sur la région cervicale. On obtient ainsi l'image d'une malformation vasculaire nourrie par deux artères artérielles : l'une supérieure née de la vertébrale, l'autre inférieure provenant d'une branche de la sous-clavière probablement la cervicale ascendante. A un temps plus tardif de gros vaisseaux veineux s'étendent tout le long du rachis cervical.

Une aortographie par voie fémorale permet de contrôler l'artère sous-clavière gauche (Fig. 2). On découvre ainsi deux nouvelles artères : l'une née de la cervicale ascendante au pôle inférieur de l'angiome, l'autre plus fine pénètre le rachis en C4—C5. Aucune artère plus bas située n'est trouvée. A un temps plus tardif le drainage veineux apparaît à partir des deux pôles de la malformation. Ce patient a subi plusieurs interventions successives destinées à exclure cet angiome du courant circulatoire. Le traitement chirurgical n'est pas encore terminé mais déjà nos contrôles angiographiques ont montré l'efficacité des ligatures artérielles : celles-ci n'ont été suivies d'aucun incident d'ordre neurologique médullaire.

Cas 3 — Femme de 34 ans. Depuis l'âge de 7 ans installation par poussées successives entre coupées de rémissions d'une paralysie complète avec troubles sphinctériens et douleurs dorsales hautes.

La myélographie lipiodolée montre un arrêt en D1 avec moulage de vaisseaux anormaux.

Les angiographies cervicales par ponction directe des artères sous-clavières droite et gauche ont montré que seule l'artère sous-clavière gauche irrigue l'angiome. L'étude angiographique est reprise quelques jours plus tard par cathétérisme rétrograde de l'artère fémorale. Le cathéter est d'abord placé dans l'artère vertébrale gauche. A un temps artériel une volumineuse artère née de l'artère vertébrale aborde l'angiome par son pôle supérieur. A un temps ultérieur s'opacifie précocement une volumineuse veine de drainage. Le cathéter est ensuite placé dans l'aorte thoracique. Une nouvelle sériographie objective une volumineuse artère inférieure née de la 7^e inter costale gauche et se divisant en deux branches qui abordent le pôle inférieur de l'angiome.

Le traitement chirurgical par temps successifs visant à exclure la malformation est actuellement en cours : il est bien supporté mais nous ne pouvons encore en fournir les résultats.

Cas 4 — Garçon de 16 ans. Une hémorragie méningée ouvre la scène clinique à l'âge de 12 ans : deux nouveaux épisodes hémorragiques surviennent un an plus tard. Une étude angiographique carotidienne bilatérale et vertébro-basilaire est négative. L'année suivante de nouvelles hémorragies méningées se produisent. Des signes neurologiques s'installent : les uns orientant vers une pathologie médullaire haute, les autres traduisant une hypertension intracranienne.

La myélographie lipiodolée montre un ralentissement de la bille opaque en D6 puis des images de moulages vasculaires en D3.

Une angiographie médullaire est réalisée par voie fémorale rétrograde. Une première injection dans l'artère sous-clavière gauche montre que ce vaisseau n'irrigue pas l'angiome. On place ensuite le cathéter dans l'aorte en regard de D5. On opacifie ainsi une malformation vasculaire dont les artères artérielles sont complexes. Du côté gauche, une grosse artère née de l'aorte monte sur le flanc gauche du rachis jusqu'au 2^e espace inter costal : elle fournit à ce niveau une branche dorso-spinale qui pénètre le rachis en D2—D3 et aboutit au pôle



Fig 5 Cas 8 Aortographie a) Temps artériel tardif. Soustraction. Opacification d'énormes dilatations veineuses en grappe emplissant le canal médullaire. Deux pédicules afférents. L'inférieur volumineux naissant de la dorso-spinale dilatée de D9 (→) formant une double courbure à concavité gauche. Le supérieur de petit calibre issu de la dorso-spinale de D8 (↔) ascendante puis formant une boucle à concavité inférieure qui l'emporte au pôle supérieur de l'angiome. b) Temps veineux. Soustraction. Le pôle inférieur de l'angiome fournit deux pédicules veineux volumineux de grande étendue. L'un médian s'élève. L'autre latéralisé se jette dans les plexus veineux latéro-vertébraux.

Le diagnostic d'angiome est porté par la myélographie. Un bilan angiographique nous est demandé avant une intervention chirurgicale sur la scoliose.

L'aortographie thoracique est faite par voie fémorale. Dès le début de l'injection s'opacifie un volumineux angiome avec shunt artério-veineux précoce, nourri par une afférence principale venue de la 1^{re} intercostale droite et par une afférence accessoire née de la 2^e intercostale. Une seconde plus tard la masse angiomateuse paraît très étendue et s'opacifient déjà les premières efférences veineuses.

À un temps tardif (Fig. 5c) les efférences veineuses sont opacifiées, étendues et complexes, elles aboutissent au plexus extrarachidien et à la veine asygos dilatée.

Le traitement chirurgical visant à exclure par ligatures successives cet angiome est actuellement en cours.

Cas 7. Fillette de 3 ans 1/2. Installation en 24 heures d'une paraplégie flasque complète avec rétention d'urine et anesthésie totale dont la limite supérieure remonte à L1. À la myélographie arrêt total du lipiodol en L1.

L'enfant est opérée sur les données de la myélographie et du bilan neurologique. Le neurochirurgien découvre une malformation qui paraît purement veineuse et dont le maximum se situe en L1.



Fig. 4. Cas 7. Aortographie par voie fémorale sonde au niveau de D₃. Soustraction a) Temps artériel Face. Deux pédicules afférents volumineux des de la face postérieure de l'aorte (4 et 6^e artères intercostales). Le shunt artério-veineux se projette en arrière de D₄—D₅—D₆ il est de structure simple et constitué par un large vaisseau serpentin. b) Temps artériel Profil. On voit les deux pédicules issus de la face postérieure de l'aorte. c) Temps veineux Face. Les veines de drainage sont remarquables par leur volume et leur extension vers le haut dans la région cervicale et vers le bas jusqu'au cône terminal. d) Temps veineux Profil. Le volumineux vaisseau spiralé s'étend du la moelle cervicale au cône terminal.

La radiographie simple du rachis montre un important élargissement des distances inter-pédiculaires en D₂—D₃—D₄—D₅.

La myélographie lipiodolée faite en radiocinématographie objective un blocage incomplet en D₃ et moule des vaisseaux anormaux que l'on voit battre dans l'huile opaque.

L'injection de la malformation est réalisée par aortographie rétrograde fémorale. Le cathéter est placé dans la crosse de l'aorte. On objective ainsi la malformation artério-veineuse. Elle commence à s'opacifier sur le premier cliché 1/2 seconde après le début de l'injection et s'étend sur les clichés pris à des temps ultérieurs de D₁ à D₅. Elle reçoit deux afférences. La plus importante par son calibre vient de la 5^e artère intercostale aortique droite. Elle pénètre le rachis en D₅—D₆ et atteint le pôle inférieur de la malformation. L'autre de moindre calibre est la branche spinale de la 3^e intercostale aortique droite qui atteint le pôle supérieur de l'angiome. Les efférences veineuses naissent très précocement. Deux supérieures importantes une inférieure grêle. Une laminectomie a révélé le siège postéro-latéral droit de l'angiome. On a montré une seule afférence artérielle postéro-latérale droite et des voies de drainage supérieures.

L'état neurologique de cet enfant est inchangé à ce jour où il poursuit sa rééducation dans une maison spécialisée.

Cas 6. Garçon de 13 ans. Installation à partir de l'âge de 2 ans par poussées successives avec rémissions d'une scoliose d'une paraplégie spasmodique avec troubles sphinctériens. Le



Fig. Cas 8 Aortographie a) Temps artériel tardif. Soustraction. Opacification d'énormes dilatations veineuses en grappe emplissant le canal médullaire. Deux pédicules afférents : l'inférieur volumineux naissant de la dorso-spinale latérale de D9 (→) formant une double courbure à concavité gauche ; le supérieur de petit calibre issu de la dorso-spinale de D8 (←→) ascendante puis formant une boucle à concavité inférieure qui l'en mène au pôle supérieur de l'angiome. b) Temps veineux. Soustraction. Le pôle inférieur de l'angiome fournit deux pédicules veineux volumineux de grande étendue : l'un médian sinueux ; l'autre latéral se jetant dans les plexus veineux latéro-vertébraux.

diagnostic d'angiome est porté par la myélographie. Un bilan angiographique nous est demandé avant une intervention chirurgicale sur la scoliose.

L'aortographie thoracique est faite par voie fémorale. Dès le début de l'injection s'opacifie un volumineux angiome avec shunt artério-veineux précoce nourri par une afférence principale venue de la 6^e intercostale droite et par une afférence accessoire née de la 8^e intercostale. Une seconde plus tard la masse angiomatueuse paraît très étendue et s'opacifient déjà les premières efférences veineuses.

À un temps tardif (Fig. 3c) les efférences veineuses sont opacifiées, étendues et complexes, elles aboutissent au plexus extra-rachidien et à la veine asygos dilatée.

Le traitement chirurgical visant à exclure par ligatures successives cet angiome est actuellement en cours.

Cas 7. Fillette de 3 ans 1/2. Installation en 24 heures d'une paraplégie flasque complète avec rétention d'urine et anisthésie totale dont la limite supérieure remonte à L1. À la myélographie arrêt total du liquodol en L1.

L'enfant est opérée sur les données de la myélographie et du bilan neurologique. Le neurochirurgien découvre une malformation qui paraît purement veineuse et dont le maximum se situe en L1.



Fig. 6. Cas 11. Aortographie par voie fémorale face. Soustraction. Augmentation de volume de la 3^e artère lombaire gauche dont la branche spinale pénètre le rachis entre L3 et L4 remonte jusqu'au bord inférieur de D11—D12 pour redescendre en regard de l'interligne L1—L2 où elle se divise en deux rameaux qui vont l'un au pôle supérieur l'autre au pôle inférieur d'un fin peloton angiomateux qui se projette en arrière de L1—L2.

Une aortographie est faite quelques semaines plus tard par voie fémorale rétrograde pour préciser les possibilités thérapeutiques. Une première injection au niveau de L1 n'opacifie pas l'angiome. Le cathéter est alors poussé jusqu'en D5. Lors de cette deuxième injection on voit sur les temps précoces (Fig. 4 a et b) deux afférences artérielles volumineuses nées des 4^e et 6^e inter costales et un shunt artério-veineux simple constitué par un large vaisseau serpentin. Deux secondes plus tard les veines de drainage sont remarquables par leur volume et leur extension en haut et en bas (Fig. 4 c et d) ainsi apparaît le large réservoir veineux sur lequel s'est arrêté le lipiodol et qu'avait découvert le neurochirurgien. Cet enfant a subi une intervention comportant la ligature et la section des 4^e et 6^e inter costales aortiques gauches. Cette intervention fut parfaitement supportée sans modification du tableau neurologique. Un mois 1/2 plus tard survient une aggravation du tableau neurologique avec paralysie des membres supérieurs qui persiste jusqu'à la mort quelques jours plus tard dans un tableau d'hémorragies profuses avec hyperthermie et collapsus terminal. Il n'y a pas eu de vérification nécropsique.

Cas 8. Homme de 25 ans. Début brutal des troubles à l'âge de 9 ans sous la forme d'une paraplégie rapidement régressive. Par la suite de nouveaux épisodes paralytiques surviennent entre coupés de rémissions. L'examen neurologique quand nous l'examinons découvre une paraplégie spasmotique, une hypoesthésie à tous les modes dont la limite supérieure est en D12.

L'étude radiologique simple du rachis révèle une scoliose à convexité gauche à sommet en D1 et un élargissement du canal rachidien en D9—D10. La myélographie lipiodolée objective un ralentissement du transit en D11 et des moultures vasculaires évidentes.

L'aortographie par voie fémorale permet d'injecter la malformation. La masse angiomateuse injectée à un temps artériel précoce reçoit deux afférences. Une principale branche dorso-spinale de la 9^e inter costale gauche aborde le pôle supérieur de l'angiome. L'autre moins importante est la branche dorsospinale de la 8^e inter costale gauche.



Fig 7 a) Vascularisation de la moelle dans son segment dorso-lombaire après injection d'une solution de baryum colloïdal dans les artères. l'écce anatomique Volu mineux artère d'Adamkiewicz qui arrive avec le 10 racine dorsale droite (Cliché emprunté à CORBIN (6)) b) Artère d'Adamkiewicz vue au cours d'une angiographie rénale chez un sujet de 29 ans

A un temps ultérieur (Fig 5a) les afférences artérielles sont encore visibles la masse angio-nateuse s'étend de D9 à D11 la séniographie nous a montré en pelotons vasculaires successifs le shunt artério-veineux qui semble siéger au pôle supérieur A un temps tardif (Fig 5b) le drainage se fait par deux longs vaisseaux de gros calibre dont l'un rejoint la veine lombaire ascendante l'autre se perd au delà des limites du cliché

La morphologie de cette malformation alimentée par deux afférences facilement accessibles jointe à l'existence d'un déficit permanent stable depuis trois ans et déjà important a permis d'envisager une intervention visant à affaiblir l'angiome par ligatures des pédicules nourriciers Cette intervention en deux temps a été bien supportée Son efficacité contrôlée angiographiquement n'a pas été complète car volontairement une des afférences a été respectée l'angiographie comme l'acte chirurgical ayant montré qu'elle fournissait l'artère du renflement lombaire Le malade a été un peu amélioré mais nous ne pouvons affirmer que cette amélioration soit à porter au crédit de l'intervention

Cas 9 Garçon de 11 ans ayant fait deux hémorragies consécutives La négativité des angiographies cérébrales un élargissement du canal rachidien en D11 et quelques discrets signes urologiques ont fait soupçonner le siége dorsal bas de l'angiome Une phlébographie rachidienne n'injette aucune malformation vasculaire Dans le même temps opératoire profitant de l'anesthésie est faite une aortographie abdominale par voie fémorale Au temps artériel de la première lombaire droite dilatée naît une artère ascendante qui aboutit au pôle supérieur d'un petit angiome intra rachidien situé en regard de D11—D12 A un temps ultérieur se dessinent l'angiome et une volumineuse efférence inférieure unique Cette disposition a été vérifiée lors de la laminectomie de décompression

Cas 10 Homme de 60 ans. Début à 58 ans par lombalgies sciatique droite, déficit du quadriceps droit. Deux ans plus tard aggravation à la suite d'une chute. L'examen neurologique en découvrant des signes périphériques et médullaires (signe de Babinski gauche) fait évoquer une atteinte du cône terminal. La P. L. montre un blocage à l'épreuve par Stockey et retire un liquide qui contient 5 éléments par ml et 1 g 70 d'albumine par litre.

Les radiographies simples montrent des images arthrosiques bilatérales. La myélographie lipiodolée objective un arrêt incomplet en regard de D12. Une première aortographie est faite qui est négative, mais de qualité photographique médiocre. L'intervention chirurgicale (laminectomie D10—D11—D12) découvre un angiome en D10—D11.

Une seconde aortographie est faite quelques semaines plus tard dans de meilleures conditions techniques. On opérise l'angiome en regard de D11. Il est nourri par une seule artère née de la première artère lombaire gauche. Les efférences sont impossibles à préciser.

Cas 11 Femme de 31 ans. À l'âge de 27 ans elle souffre de sciatique gauche. Quatre ans plus tard installation brutale de douleurs radiculaires thoraciques basses et d'une paraplexie avec rétention d'urine. Le diagnostic d'angiome est porté par la myélographie lipiodolée. Une phlébographie trans-épineuse est négative. Une laminectomie de décompression est faite sans résultat sur les signes neurologiques.

La malade nous est confiée ultérieurement pour préciser les connexions vasculaires de l'angiome.

Une aortographie thoraco-abdominale par voie fémorale est réalisée. Sur un temps artériel tardif (Fig. 6) on voit naître de la 3^e artère lombaire gauche dilatée une artère à trypet intrarachidien descendant qui après une boucle en regard de D12 se divise en deux rameaux qui vont l'un au pôle supérieur, l'autre au pôle inférieur d'un fin peloton angiomateux. L'image angiographique recoupe l'aspect vu à l'intervention.

Cas 12 Homme de 43 ans. Début à 40 ans par des douleurs dans le membre inférieur gauche un an plus tard parapésie et hypoesthésie globale remontant jusqu'à D12.

La notion d'un angiome cutané lombaire opéré antérieurement jointe à la constatation d'un arrêt de lipiodol en arrière de L2 font pratiquer en juin 1962 une laminectomie qui découvre une malformation vasculaire inextirpable au niveau du cône terminal.

À la suite de cette intervention une récupération s'amorce mais 8 mois plus tard survient une aggravation.

Le malade nous est alors adressé pour exploration angiographique. L'aortographie par voie fémorale objective un anévrysme artério-veineux de petit volume nourri par une artère unique née de la 11^e intercostale thoracique gauche représenté par un fin peloton vasculaire en regard de D12 et L1 drainé par une efférence née du pôle inférieur de la malformation.

Discussion

Toutes ces observations démontrent que le diagnostic de malformation vasculaire médullaire est possible par l'angiographie. Notre intention n'est pas de nier l'intérêt diagnostique de la myélographie lipiodolée, cependant, l'angiographie a le mérite d'apporter une preuve directe formelle. Ce diagnostic a reçu onze fois une confirmation anatomique opératoire ou nécropsique.

Precisons que nous n'avons jamais observé de complication en particulier, aucune modification de l'état neurologique antérieur du patient, au décours de ces explorations vasculaires.

L'angiographie permet en outre, un bilan exact de la malformation, elle objective les afférences artérielles le siège du ou des shunts artério-veineux et les efférences veineuses. Elle montre souvent d'ailleurs une lésion dont le shunt et l'extension le long de la moelle sont plus complexes que ne le laissait supposer la myelographie et même l'exploration chirurgicale. Notre observation n° 7 en est une parfaite illustration.

Ainsi l'angiographie permet une plus claire vision de la lésion. À ce point de vue elle facilite l'acte opératoire car elle permet au chirurgien de mieux comprendre la systématisation des vaisseaux anormaux qu'il découvre.

Cette meilleure étude anatomique due à l'angiographie permet quelques considérations nosologiques.

Dans tous les cas que nous avons examinés, il s'agissait de malformations artério-veineuses avec shunt. Nous n'avons jamais retrouvé chez ces malades réunis sans sélection préalable, la classique différenciation entre angiomes artériels, angiomes artério-veineux, angiome racémeux veineux. Bien au contraire, nous trouvons une disposition angiographique absolument calquée sur celle de ces malformations au niveau de l'encéphale.

Considérons tout d'abord les afférences artérielles.

Les artères aboutissant à la malformation sont des artères normales par leur origine, leur trajet, leur mode de pénétration dans le rachis, la concordance topographique de leur niveau d'origine et des segments médullaires intéressés.

Ces artères sont anormales par l'augmentation de leur calibre, témoin de l'absence de barrière capillaire et meilleur garant de leur visualisation arteriographique. Elles sont anormales par leur mode de terminaison au sein d'un réseau vasculaire vestigial court circuitant artère et veine.

Considérons en second lieu la masse angiomateuse.

Elle est toujours difficile à analyser. La scintigraphie rapide permet le plus souvent de reconnaître sur les temps précoces, le cœur de la malformation, le siège principal des fistules artério-veineuses. Rapidement la superposition noie dans une même opacité afférences dans leur segment terminal, vaisseaux angiomateux et efférences. Le volume de l'angiome est très variable selon sa topographie, nous y reviendrons plus loin. Son siège par rapport à la moelle est difficile à préciser, dans un certain nombre de cas où l'on a pu varier les incidences, on peut affirmer le siège principalement postérieur des vaisseaux anormaux qui parait la règle.

Considérons enfin les efférences.

Elles sont variables en nombre comme en volume et difficilement systéma-

utiles. Le fait le plus frappant est qu'elles forment quant à l'espace occupé la partie la plus importante de la malformation dans un grand nombre de cas. Ceci s'explique par la dilatation variqueuse des veines qui se poursuit très loin en aval du shunt artério-veineux, dépassant en haut et en bas l'angiome proprement dit, pour intéresser parfois l'ensemble de la moelle comme dans notre observation n° 7.

Ces constatations permettent de comprendre les erreurs fréquemment commises dans l'appréciation du volume, de la topographie et de la nature même de ces malformations, telle qu'elle est faite lors des interventions chirurgicales ou des vérifications anatomiques. On conçoit ainsi qu'une exploration chirurgicale limitée puisse conclure à un angiome purement veineux si le shunt artério-veineux lui-même et les artères artérielles sont distantes de la région explorée. Certains travaux récents tel celui de ANTONI (2), aboutissent aux mêmes conclusions que nous sur la base d'études anatomiques sérieuses. Le grand mérite de l'angiographie est de clarifier jusqu'à l'évidence la structure complexe de ces lésions en visualisant successivement leurs différentes composantes.

Si l'on étudie la malformation dans son ensemble et si l'on veut tenter une systématisation, autant que cela soit permis sur une série restreinte de cas, il semble que l'on puisse distinguer deux types différents.

1) Les fistules artério-veineuses simples, siégeant électivement sur l'extrémité inférieure de la moelle, alimentées par une artère artérielle unique, représentées par un peloton artério-veineux de petit volume et drainées par une seule efférence veineuse (4 cas dans notre série).

2) Les anévrysmes circoïdes pour lesquels les artères artérielles sont multiples, la masse angiomatueuse volontiers volumineuse, les efférences veineuses importantes et complexes. C'est ainsi que se présentent les angiomes cervicaux (4 cas) et dorsaux (4 cas).

Cette distinction recoupe en la précisant la séparation en malformations focales et pluri segmentaires suggérée par BRION, NETSCHI & ZIMMERMAN (4).

Les limites diagnostiques de l'angiographie médullaire sont encore difficiles à préciser compte tenu des 12 cas positifs que nous rapportons, de 3 cas douteux qui n'ont pas été vérifiés et n'ont pas été inclus dans cette statistique et de nos connaissances actuelles des images angiographiques normales des vaisseaux médullaires. Ces références angiographiques normales sont en effet difficiles à obtenir. Nous en avons commencé l'étude, pour le segment terminal, en examinant soigneusement nos dossiers d'angiographies rénales. Lorsque nous la voyons sur les films (Fig. 7a), l'artère du renflement lombaire ou artère de ADAM KIEWICZ (1) a toujours la même morphologie, celle qu'ont révélée les études anatomoradiologiques sur moelle préparée (Fig. 7b) née d'une intercostale

inférieure ou d'une lombaire supérieure cette artère pénètre dans le rachis où elle décrit d'abord un trajet ascendant vers la ligne médiane puis une boucle en épingle à cheveux enfin un trajet verticalement descendant du sommet de la boucle se détache une fine artériole ascendante. On découvre parfois de fines artères médullaires accessoires. Nous n'avons pas encore terminé cette étude anatomique pour le renflement lombaire il nous est impossible pour l'instant de préciser la fréquence avec laquelle cette artère est accessible à l'angiographie chez le sujet normal non plus les facteurs techniques capables de nous fournir d'une façon plus fréquente cette image.

Il nous faut envisager également les problèmes de diagnostic différentiel qui vont surgir du fait de l'utilisation de ces nouvelles méthodes. Les anévrysmes artériels purs sont exceptionnels au niveau de la moelle, quelques cas en ont été cependant décrits tel celui rapporté par LOMBARDI & MURELLO (26) il est probable que ces anévrysmes dans la mesure où ils ne sont pas thrombosés sont accessibles à l'angiographie.

Les tumeurs intra rachidiennes quand elles sont de nature vasculaire, hémangioblastomes, angioréticulomes, hémangioépécytomes sont également susceptibles de s'opacifier par voie artérielle. DI CHIRO (8) a rapporté en 1957 une observation de maladie de von HIPPEL LINDAU à double localisation cérébrale et cervicale supérieure cette dernière localisation était parfaitement mise en évidence par l'angiographie.

Enfin il est probable que les autres processus tumoraux intra rachidiens lorsqu'ils ont richement vascularisés peuvent avoir eux aussi une traduction angiographique.

Pour notre part nous pensons qu'un large champ est ouvert à l'angiographie médullaire. Ce sera là le but de nos recherches dans les années à venir.

Pour terminer soulignons que dans notre esprit l'intérêt de ces explorations n'est pas seulement iconographique et diagnostique. Pour la première fois dans le domaine des angiomes de la moelle, à côté de la chirurgie palliative ou aveugle pratiquée jusqu'à maintenant elles ouvrent la voie à des solutions chirurgicales nouvelles.

Elles permettent d'envisager soit des interventions d'isolement de ces malformations par ligature de leurs afférences soit sous certaines conditions techniques particulières un abord direct de certaines de ces lésions en bonne connaissance de leurs connexions anatomiques. Cinq de nos patients ont été soumis à des tentatives chirurgicales de cet ordre.

Il est encore trop tôt pour tirer des conclusions sur la valeur de ces techniques chirurgicales nouvelles. Nous savons combien ces problèmes sont complexes, il nous faudra un plus grand nombre de cas, un recul plus important dans le temps avant que nos neurochirurgiens puissent faire un bilan valable.

RÉSUMÉ

La vascularisation de douze cas de malformation vasculaire de la moelle à localisations cervicales thoraciques ou dorso-lombaires a été étudiée par angiographie. Il est possible, actuellement, au prix de précautions techniques simples, d'objectiver, comme pour les malformations vasculaires cérébrales, les afférences, les éfférences, les shunts et les éfférences. Dans tous les cas étudiés, il s'agissait d'angiomes artério-veineux avec shunt.

SUMMARY

The vascularisation in twelve cases of vascular malformation of the spinal cord situated in the cervical, thoracic or dorsolumbar regions was studied by angiography. Observing simple technical precautions, it is possible, as in the case of cerebral vascular malformation, to demonstrate afferent and efferent vessels, as well as shunts. All cases studied had arteriovenous angiomas with shunt.

ZUSAMMENFASSUNG

Es wurde in 12 Fällen die Gefäßversorgung von vaskulären Missbildungen des Rückenmarks der cervicalen, thorakalen oder lumbodorsalen Region mittels Angiographie studiert. Unter Einhaltung einfacher technischer Vorsichtsmaßnahmen ist es möglich, bei den Fällen mit cerebralen vaskulären Missbildungen, afferente und efferente Gefäße wie auch Shunts nachzuweisen. In allen untersuchten Fällen handelte es sich um arteriovenöse Angiome mit Shunt.

BIBLIOGRAPHIE

1. ADAMIEWICZ A. Die Blutgefäße des Menschlichen Rückenmarkesoberfläche. S. B. Akad. Wiss. Wien math. nat. Kl. 85 (1882) 101.
2. ANTONI N. Spinal vascular malformations (angiomas) and myelomalacia. *Neurology* 19 (1962) 795.
3. ARSENI C. and SAMITCA D. Vascular malformations of the spinal cord. *Acta psychiat. scand.* 34 (1959) 10.
4. BRION S., NETZKY M. and ZIMMERMAN H. Vascular malformations of the spinal cord. *A. M. A. Arch. Neurol. Psychiat.* 68 (1952) 339.
5. BUCHANAN D. and WALKER M. Vascular anomalies of the spinal cord in children. *Amer. J. Dis. Child.* 61 (1941) 928.
6. CORBIN J. L. Anatomie et pathologie artérielles de la moelle. Masson Ed. Paris 1961.
7. CUSHING H. and BAILEY P. Tumors arising from the blood vessels of the brain. Ch. Thomas, Baltimore 1928.
8. DI CHIRO G. Combined retino cerebellar angiomatosis and deep cervical angiomas. *J. Neurosurg.* 14 (1957) 685.
9. DJINDJIAN R., DUMESNIL M., FAURÉ C., LEFEBVRE J. et LAVEQUE B. Etude angiographique d'un angiome intra rachidien. *Rev. neurol.* 106 (1962) 278.
10. — — — et TAVERNIER C. Angiome medullaire dorsal. Etude clinique et artériographique. *Rev. neurol.* 108 (1963) 432.

- 11 — HILDART R, LEFEBVRE J, LE BESVERAIS Y et HURTH M. L'artériographie des angiomes de la moelle (Intérêt nosographique et perspectives thérapeutiques) *Rev neurol* 109 (1963) 640
- 12 EPSTEIN J, BELLER A and COHEN J. Arterial anomalies of the spinal cord. *J Neurosurg* 4 (1949) 45
- 13 CARCIN R, ZULCH K J, LAZORTHES G et GRUNER J. Pathologie vasculaire de la moelle. *Rev neurol* 106 (1962) 531
- 14 GINSBOURG M. Contribution à l'étude des formations vasculaires pathologiques vertébro-médullaires (angiomes vertébraux épидурaux intraduraux). Thèse Les Deux Artisans Ed. Paris 1961
- 15 GRAVELEAU D et PADILETTI C. Malformations et tumeurs vasculaires de la moelle chez l'enfant: à propos de deux observations. *Ann Pediat* 38 (1963) 310
- 16 GUILLAIN G et ALAJOUANINE TH. Paraplégie par compression due à un volumineux angiocèle de la pie mère spinale. Contribution à l'étude des compressions médullaires dues à des formations vasculaires pathologiques. *J Neurol (Brux)* 11 (1975) 69
- 17 GLIOT G, HILDART R et HILDART F. Les malformations vasculaires angiomatueuses de la moelle. *Sem Hop Paris* 22 (1946) 809
- 18 HENSON R A and CROFT P B. Spontaneous spinal subarachnoid hemorrhage. *Quart J Med* 25 (1956) 53
- 19 HOOK O and LIDVALL H. Arteriovenous aneurysms of the spinal cord. *J Neurosurg* 15 (1958) 84
- 20 HILDART F. Contribution à l'étude des angiomes de la moelle. Angiomes intra-durax extramédullaires. Thèse Paris 1944
- 21 HURTH M. Les anévrismes artério-veineux de la moelle épinière. Considérations anatomocliniques et thérapeutiques à propos de onze cas étudiés par artériographie. Thèse Foulon Ed. Paris 1964
- 22 LAZORTHES G et coll. La vascularisation artérielle de la moelle. Recherche anatomiques et applications à la pathologie médullaire et à la pathologie aortique. *Neuro-chirurgie* 4 (1958) 3
- 23 LEFEBVRE J, DJINDJIAN R, DUMESNIL M et FAURÉ C. Angiomes intrarachidiens et objets étudiés par angiographie. *Ann Radiol* 6 (1963) 165
- 24 LOVBARDI G. Studio radiologico degli angiomi spinali. *Radiol m d (Torino)* 37 (1951) 716
- 25 — and PASSERINI A. Spinal cord tumors. *Radiology* 76 (1961) 381
- 26 — et MORELLO C. Causes rares d'élargissement du canal rachidien. *Acta radiol* 50 (1958) 230
- 27 — and MIGLIUACCA F. Angiomas of the spinal cord. *Brit J Radiol* 32 (1959) 810
- 28 MORRIS L. Angioma of the cervical spinal cord. *Radiology* 75 (1960) 783
- 29 NEWMAN M J III. Racemose angioma of the spinal cord. *Quart J Med* 28 (1959) 97
- 30 RAND R W and RAND C W. Intraspinal tumors of childhood. Charles C Thomas Springfield U.S.A. 1960
- 31 SADA K and MERRIT A A. Vascular anomaly of the spinal cord illustrated by three case reports. *Aust Ann Med* 5 (1956) 136
- 32 SELDINGER J I. Catheter replacement of the needle in percutaneous arteriography. *Acta radiol* 39 (1953) 368
- 33 SYTEN H T and BAKER H L. Roentgenography and surgical aspects of the vascular anomalies of the spinal cord. *Surg Gynec Obstet* 112 (1961) 729
- 34 TARLOW I M. Spinal extradural hemangioblastoma roentgenographically visualised with diodrast at operation and successfully removed. *Radiology* 49 (1947) 717

- 35 WALKER A. T. Dilatation of vertebral canal with congenital anomalies of the spinal cord. *Amer J Roentgenol* 52 (1944) 571
- 36 WILBURN MASSON. The vascular abnormalities and tumors of the spinal cord and its membranes. H. Kimpton Ed. London 1943
- 37 ZIRDSER DES PLANTES II. Application of the roentgenographic subtraction method in neuroradiology. *Acta radiol* 1 (1963) 961

TOTAL ANGIOGRAPHY IN SPONTANEOUS INTRACRANIAL HAEMORRHAGE

by

A. A. DONALDSON

The problem of early location of the site of bleeding in spontaneous intracranial haemorrhage has been a major concern in our department for many years. For seven years it has been our practice to carry out a complete survey of the cerebral circulation in cases of spontaneous intracranial haemorrhage. Initially this survey was carried out by individual injection of each carotid artery and each vertebral artery the usual sequence followed being a) bilateral carotid angiography, b) 24—48 hours later right vertebral angiography, c) a further 24—48 hours later left vertebral angiography. If the clinical features of the case made it necessary the sequence was modified to suit.

In 1959 the author reported to the Scottish Radiological Society a first series of 25 consecutive cases of total cerebral angiography (DONALDSON 1959) in which an aneurysmal sac was demonstrated at the origin of the posterior inferior cerebellar artery on the fourth vessel investigated in 4 cases. In none of these cases was the clinical syndrome of localising value. All the cases were submitted to operative treatment and in each the radiological appearances were confirmed.

The delay in making a definitive diagnosis caused us much anxiety because of the risk of further perhaps catastrophic haemorrhage and following on our

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Fig 1 Total cerebral angiography Axillary catheter Aneurysm on right middle cerebral artery

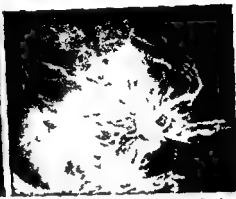


Fig 2 Total cerebral angiography Plethora of vessels in lateral view No aneurysmal sac seen in this and supplementary views

30 to 40 ml of contrast medium are injected by means of a pressure injector. The Talley pump is favoured but other similar devices are suitable. Posterior, lateral and axial views are normally obtained. Other views, particularly an oblique view, may also be helpful. In view of the plethora of vessels shown, stereo lateral views are probably advisable.

Where doubtful appearances are encountered, supplementary individual carotid or vertebral injections can be carried out, either at the same session or subsequently. Apart from the fatality with the method now discarded for this particular type of case, only 1 transient left hemiparesis has occurred in the 76 patients investigated. This was in a case where retrograde brachial injection was used; recovery took place within a few hours, leaving no residual disability. There has been no serious circulatory impairment of the limbs following catheterisation. In one case of femoral catheterisation, however, a fairly large haematoma became evident some hours following the procedure.

In two cases, because of very tortuous or atheromatous vessels, the catheterisation was unsuccessful (1 femoral and 1 axillary) and the examinations were completed in the standard manner using carotid and other injections. Although the axillary route is preferred for the catheter, in four of our early cases some difficulty was experienced in passing the catheter beyond the branches of the innominate and femoral catheterisation was carried out instead. In the case of difficulty with either route, it is worth while to proceed immediately to the other. We have not encountered this difficulty in our more recent cases, using a catheter with a suitably curved tip.

Of the 76 examinations, 9 were post-operative control. These had mostly undergone therapeutic carotid ligation and were important in that vertebral

studies of obstructive cerebral vascular disease by catheterisation of the aortic arch via the femoral artery, it was felt that this provided a means for a rapid preliminary survey of the whole cerebral circulation at the earliest possible time, especially in cases where the clinical syndrome was unhelpful in localisation.

The series here reported totals 76 cases. Femoral catheterisation was used in 39 cases and carotid catheterisation in 28 cases. Of the remaining 9 cases, 7 were subjected to retrograde brachial artery injection by the technique described by KUHN & KUGLER (1963) and the remaining 2 according to the carotid injection technique of ZAGLIS (1959).

Although this latter technique produces excellent filling of the intracranial vessels, we consider that the fall in blood pressure that occurs, although present for a very brief time, is too serious for the method to be applied routinely in acute cases of spontaneous intracranial haemorrhage. The only death subsequent to angiography in the present series occurred after employing this method, and autopsy revealed widespread infarction in both cerebral hemispheres. There was an unusually widespread hyaline necrotising arteritis. No aneurysm or other vascular anomaly was found.

Cannulation of the right brachial artery produces excellent filling of the right carotid and vertebral circulations and in 10 % of cases the left carotid circulation is also filled. With left carotid occlusion in 50 % of cases the left carotid circulation is also filled. These figures are according to KUHN & KUGLER and our experience is similar. An important disadvantage is that the upper portion of the left vertebral artery with its posterior inferior cerebellar branch are not usually shown. This, in our view, necessitates further left brachial or left vertebral injection.

Some radiologists consider that the need to expose the brachial artery is a disadvantage, but we find this no problem. The need to carry out supplementary left carotid angiography either by retrograde left temporal injection or direct carotid injection is probably a disadvantage.

Total angiography by carotid or femoral catheterisation appears to be a simple and safe procedure. The majority of the examinations in the present series, i.e. 67 out of 76, have been carried out using this method. The standard Seldinger technique using the large bore needle and a grey Kofka catheter has been employed in adults. The carotid route is preferred as this allows the use of a shorter catheter and permits a more rapid injection of contrast medium. The newer, less viscous, contrast media, such as sodium metrizoate (Triosil) in 60 % or 75 % concentration, or sodium iothalamate (Angio Conray 80) are now used routinely. These less viscous media have greatly improved the contrast obtainable in the intracranial vessels.

For each series of films using the Schonander rapid serial film changer,



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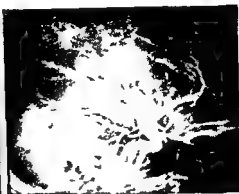


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SUMMARY

Despite a relatively large proportion of negative findings total cerebral angiography using mainly femoral or axillary catheterisation has proved useful for the localisation of acute spontaneous intracranial haemorrhage. Technical aspects of the procedure are discussed.

ZUSAMMENFASSUNG

Trotz einer ziemlich grossen Anzahl negativer Resultate hat sich totale Zerebralangiographie wobei hauptsächlich femorale oder axiale Katheterisierung verwendet wird für die Lokalisierung von spontanen akuten intrakraniellen Hämorrhagien von Wert gezeigt. Technische Aspekte dieses Verfahrens werden diskutiert.

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Malgré une proportion relativement importante d'examen négatifs l'angiographie cérébrale totale par cathétérisme fémoral ou axillaire a été montrée utile pour la localisation des hémorragies intracranéennes aiguës spontanées. L'auteur étudie certains aspects techniques de cet examen.

REFERENCES

- DONALDSON A. A. Bilateral vertebral angiography. Proc. of the Scottish Radiological Society 1959.
- KLIN R. A. and KUGLER H. Angiographic methods of choice in evaluation of stroke patients. Bull. Johns Hopk. Hosp. 119 (1963) 59.
- ZACLES J. A new method of cerebral angiography. Arch. Neuro-psiquiat. (S. Paulo) 17 (1959).
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Of the 76 cases, 36 gave positive findings of aneurysm or arteriovenous malformation. Most cases had a single aneurysm which was considered responsible for the intracranial haemorrhage, but 4 cases had multiple aneurysms, two with two aneurysms, one with three aneurysms, and one with a total of five aneurysms. There was one basilar aneurysm, and the series is completed by the demonstration of five arteriovenous malformations. In 16 of the positive cases, further examination was carried out to obtain further information but this figure of 16 includes a few post operative control examinations following surgery.

The negative examinations, i.e. negative for aneurysm or other vascular malformation, were 40 in number. Degenerative vascular disease as shown by atheroma or tortuosity of the vessels has not been included as a positive finding although in a number of cases this was present. About one third of the negative cases, namely 14, were further examined by carotid and/or vertebral angiography and this invariably tended to confirm the original negative findings. The remainder of the negative cases (22) were subjected to further angiography. Practically all the negative cases were examples of a minor intracranial haemorrhage without focal signs. Some of the patients were hypertensive. These patients have been followed up clinically only for periods up to about 18 months. No recurrent haemorrhage has been reported except in one case which did in fact show an anterior communicating aneurysm. At the time of the original total angiography, both the surgeon in charge of the case and the author classified this as doubtful and requiring further investigation, but the patient, a 61 year old diabetic woman with severe generalised arterial disease, hypertension and retinitis, died of recurrent bleeding before further angiography was carried out, i.e. within 48 hours. This we feel may have been an error of judgement as, in retrospect, there is no doubt about the presence of the aneurysm (confirmed at autopsy). The patient was, of course, a very poor operative risk.

The significance of the figures is difficult to assess at this stage. The numbers are small for adequate statistical analysis and the series is complicated by the fact that during the earlier period of the study other cases of spontaneous intracranial haemorrhage which had a definite focal syndrome were investigated by conventional carotid and vertebral angiography. At this time, total angio-

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REFERENCES

- DONALDSON, A. A. Bilateral vertebral angiography. *Proc. of the Scottish Radiological Society* 1959.
- KUHN, R. A. and KUGLER, H. Angiographic methods of choice in evaluation of stroke patients. *Bull. Johns Hopk. Hosp.* 112 (1963) 59.
- ZACLES, J. A new method of cerebral angiography. *Arch. Neuro-psiquiat. (S. Paulo)* 17 (1959).
- and CRUZ, O. R. Arterial occlusions of carotido-vertebral system. *Arch. Neuro-psiquiat. (S. Paulo)* 19 (1961) 293.

angiography had not previously been carried out. No further aneurysms were found. Three of these cases had undergone treatment for arteriovenous malformation and one in particular was very important in that a saccular aneurysm on the middle cerebral circulation which had been suspected at the original examination was excluded by total angiography. In all cases it was felt that the examination was satisfactory and useful.

Of the 76 cases, 36 gave positive findings of aneurysm or arteriovenous malformation. Most cases had a single aneurysm which was considered responsible for the intracranial haemorrhage, but 4 cases had multiple aneurysms, two with two aneurysms, one with three aneurysms, and one with a total of five aneurysms. There was one basilar aneurysm, and the series is completed by the demonstration of five arteriovenous malformations. In 16 of the positive cases, further examination was carried out to obtain further information but this figure of 16 includes a few post-operative control examinations following surgery.

The negative examinations, i.e. negative for aneurysm or other vascular malformation, were 40 in number. Degenerative vascular disease as shown by atheroma or tortuosity of the vessels has not been included as a positive finding although in a number of cases this was present. About one third of the negative cases, namely 14, were further examined by carotid and/or vertebral angiography and this invariably tended to confirm the original negative findings. The remainder of the negative cases (22) were subjected to further angiography. Practically all the negative cases were examples of a minor intracranial haemorrhage without focal signs. Some of the patients were hypertensive. These patients have been followed up clinically only for periods up to about 18 months. No recurrent haemorrhage has been reported except in one case which did in fact show an anterior communicating aneurysm. At the time of the original total angiography, both the surgeon in charge of the case and the author classified this as doubtful and requiring further investigation, but the patient, a 64 year old diabetic woman with severe generalised arterial disease, hypertension and retinitis, died of recurrent bleeding before further angiography was carried out, i.e. within 48 hours. This we feel may have been an error of judgement as, in retrospect, there is no doubt about the presence of the aneurysm (confirmed at autopsy). The patient was, of course, a very poor operative risk.

The significance of the figures is difficult to assess at this stage. The numbers are small for adequate statistical analysis and the series is complicated by the fact that during the earlier period of the study other cases of spontaneous intracranial haemorrhage which had a definite focal syndrome were investigated by conventional carotid and vertebral angiography. At this time, total angio-



Fig 1 Thombosis of internal carotid artery with filling of anterior circulation through a large posterior communicating artery. The only neurologic deficit was an upper visual field defect in the right eye.

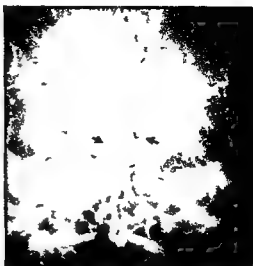


Fig 2 Bilateral vertebral arteriogram by simultaneous injection of the subclavian arteries. Filling of all the cerebral arteries in both hemispheres from the posterior circulation through the circle of Willis. No residual neurologic deficit but recurrent episodes of aphasia and right hemiparesis.

frequently that they cannot always be attributed to extension of clot into peripheral branches or to detachment of emboli. This recurrent loss of function apparently on an ischemic basis seems more likely to be related to a temporary failure of the compensatory mechanisms involved in collateral circulation.

Such intermittent attacks of transitory paresis or paresthesia may continue without change over many months or years, recurring at irregular intervals. In a considerable number of individuals they may be forerunners of a permanent neurologic deficit. Such a catastrophic event is usually related to the occurrence of complete occlusion of the primary vessel and coincident failure of collateral circulation to compensate for the cessation of flow through the main channel. During the period of partial obstruction there is a continuing effort on the part of both the primary vessel and the collaterals to supply the territory of the peripheral branches. When complete occlusion of the primary channel occurs the struggle ceases. This can result in irreversible deficit if the compensatory mechanisms in collateral circulation are inadequate or in reestablishment of a homeostatic situation if they are adequate. In the latter circumstance the previously recurring transient ischemic attacks may cease abruptly and the patient remain free of symptoms. In some cases

COLLATERAL CIRCULATION IN EXTRACRANIAL VASCULAR OCCLUSION

by

WILLIAM S. FIELDS

Angiography has become one of the most important steps in the diagnosis of occlusive vascular disease. This technique has contributed much needed information, otherwise unobtainable, regarding the significance of collateral circulation. This is especially apparent in respect to understanding two separate but related aspects of cerebral ischemia: 1) the pertinent clinical manifestations of vascular occlusion, and 2) the variability of recovery from functional neurologic deficit.

Angiographic studies demonstrate that complete occlusion of one internal carotid artery or one vertebral artery does not always result in permanent functional deficit in neural structures, as has been frequently stated in the past. Even complete obstruction of both internal carotids, of one carotid and one vertebral, or of both vertebral arteries may not produce marked disability when compensatory mechanisms can immediately become operative.

Before occlusion becomes complete, and while stenosis is gradually becoming more severe, the collateral circulation may not adjust rapidly enough to prevent transient episodes of ischemia when alterations occur in the systemic circulation. Functional loss during these attacks can be quite severe, but rapid recovery usually follows without residual disability. The episodes may recur so



Fig 1 Thrombosis of internal carotid artery with filling of anterior circulation through a large posterior communicating artery. The only neurologic deficit was an upper visual field defect in the right eye.



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Fig. 3 Occlusion in proximal portion of the vertebral artery. distal portion is filled through numerous collaterals originating from the ipsilateral thyrocervical trunk.



Fig. 4 Occlusion of the basilar artery in a patient with a history of recurrent episodes of basilar insufficiency. Flow from the anterior into the posterior circulation through the circle of Willis. Superior cerebellar arteries filled in retrograde direction.

where collateral circulation has failed to develop, the initial manifestation of occlusive disease may be catastrophic without antecedent history of recurring transitory episodes.

Satisfactory diagnostic evaluation of patients with cerebrovascular insufficiency requires complete demonstration of the arterial tree from the origin of the great vessels in the aortic arch to the intracranial branches of the cerebral arteries. It is exceedingly important to have information regarding anomalies, which are very frequent in this part of the circulation. It is well known that morphologic variation is common in the circle of Willis and that the integrity of this vascular configuration is of considerable significance in determining whether a patient with cerebrovascular disease will have permanent residual deficit. It is for this reason that any study of the cranio-cervical vessels must include adequate views to outline the circle of Willis.

Occlusive lesions are most common at points of bifurcation of the major arteries. When thrombosis occurs, the parent vessel becomes occluded, distally at least, to the next point of branching, resulting in classical patterns of colla-

teral circulation which differ for each of the major arterial channels. Many of these have been described by anatomists in the dissecting room but they can be readily observed angiographically if time and effort are devoted to obtaining adequate films.

In view of the increasing interest during the past decade in the surgical treatment of extracranial arterial occlusion it has become much more important to study these patients carefully since decisions regarding operability and prognosis are more readily arrived at if proper angiographic demonstration is obtained.

Some examples are presented of angiograms from patients with cerebrovascular disease with either no deficit or minimal residual neurologic deficit (Figs 1 to 4). It will be apparent from the material presented that the presence or absence of disability is dependent upon the adequacy of collateral circulation.

SUMMARY

Roentgenographic demonstration of the cranio-cervical collateral circulation has contributed to the understanding of the protean clinical manifestations of arterial occlusion and the variability of recovery from functional deficit. Transient attacks of cerebral ischemia cannot always be attributed to emboli but are frequently related to temporary failure of compensatory mechanisms involved in collateral circulation. Satisfactory diagnostic evaluation requires complete demonstration of the arterial blood supply to the brain. Angiograms are presented from studies of patients with advanced occlusive disease and little or no clinical deficit.

ZUSAMMENFASSUNG

Der rontgenologische Nachweis der kranio-cervikalen Kollateralzirkulation hat zum Verstehen der verschiedenen klinischen Manifestationen von arteriellem Gefäßverschluss und der Verschiedenartigkeit der Vorgänge bei Wiederherstellung von funktionellen Störungen beigetragen. Vorübergehende Anfälle von cerebraler Ischämie können nicht immer auf Embolie zurückgeführt werden, stehen jedoch häufig in Beziehung zu zeitweiligem Versagen von kompensatorischen Vorgängen der Kollateralzirkulation. Zufriedenstellende diagnostische Klärung erfordert vollständige Darstellung der art Blutversorgung des Gehirns. Es werden Angiogramme von Patienten mit mäßigem Gefäßverschluss und geringen oder gar keinen klinischen Ausfallserscheinungen gezeigt.

RÉSUMÉ

La mise en évidence radiologique de la circulation collatérale cranio-cervicale a contribué à faire comprendre les manifestations cliniques protéiformes de l'obstruction artérielle et la variabilité de la récupération des déficits fonctionnels. Les crises transitoires d'ischémie cérébrale ne peuvent pas toujours être imputées à des embolies mais sont souvent dues à une défaillance temporaire de mécanismes compensateurs qui interviennent dans la circulation collatérale. Un bilan diagnostique satisfaisant nécessite la mise en évidence de toutes les artères afférentes au cerveau. Les auteurs présentent des angiographies de malades atteints d'obstructions artérielle avancées manifestant par un déficit clinique minime ou nul.



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REFERENCES

- FIELDS W. S. and WFIBEL J. Effects of vascular disorders on the vestibular system. In Neurological aspects of auditory and vestibular disorders. Eds., W. S. Fields and B. R. Alford. Charles C. Thomas, Springfield, Ill. 1964.
- BRUTTMAN M. L. and WFIBEL J. Collateral circulation of the brain. Williams & Wilkins, Baltimore, Maryland, U.S.A. (In press).
- WFIBEL J., FIELDS W. S., BRUTTMAN M. I., CRAWFORD F. S. and KIRCHHOFF P. C. Pneumarteriographie der intra- und extrakraniellen Zirkulation (Ergebnisse mit perkutaner Subclavia-Katheterisation). Fortsch. Röntgenstr. 99 (1963), 354.
- — Direct percutaneous infracranial catheterization of the subclavian artery. J. Neurosurg. 20 (1963), 233.

LOCALISATION OF THE VENOUS ANGLE WITH CONSIDERATION PAID TO THE SIZE AND SHAPE OF THE CRANIAL VAULT

by

ERICH FISCHER

Since the first method for localisation of the venous angle of the deep cerebral veins was published in 1951 9 further methods or improvements have become known. The multitude of methods proves that there is not yet a sufficiently exact localisation method meeting practical requirements.

After examining the existing methods it became clear that an improvement could only be obtained if the individual size and shape of the cranial vault was taken into consideration by a suitable system of coordinates (FISCHER 1963).

The systematic search for a suitable system of coordinates was based on 200 lateral phlebograms.

1 Almost all lines and angles between main anatomic points situated in the midline of the cranial vault and of the skull base have been examined with respect to their correlative dependency.

2 The angles as demonstrated in Fig. 1 of which one side intersects with the venous angle respectively have been checked with respect to their mutual correlation and their correlation to cardinal skull indices such as height length index and angle of the skull base.

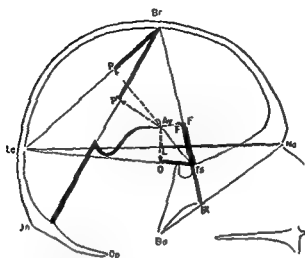


Fig. 2 The vertical from venous angle to lines Br Ts Br Bo Br La Br In Na La and Ts La. The ratio of the heavily drawn section to the total line has been calculated

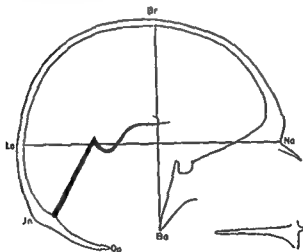


Fig. 3 The venous angle always lies above the line Na La and on the average on the line Br Bo

Furthermore vertical lines have been erected in the venous angle on 6 lines as shown in Fig. 2. The ratio of the section to the total line has been determined and this again was correlated with a multitude of data.

A simple approximative as well as an exact method for localisation of the

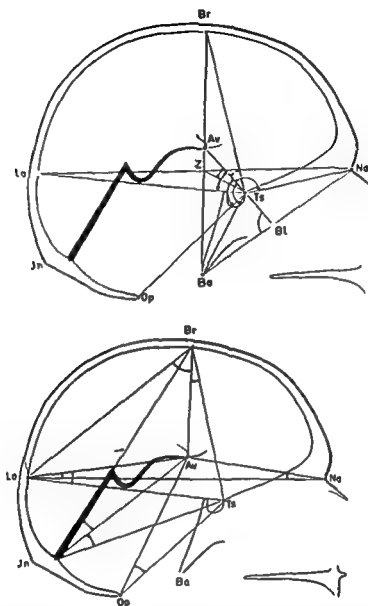


Fig 1 Angle between angulus venosus and points of the bony skull with angular point at the skull base (top sketch) and at the cranial vault (bottom sketch) Diagrams constructed according to medium values taken from 200 normal cerebral phlebograms of adults Na = nasion Br = endobregma La = endolambda In = endinion Op = opisthion Ba = basion Ts = tuberculum sellae Av = venous angle Z = central point of skull Bl = extension of line Av Tr to base line Na Ba (top sketch) respectively extension of line Br Ts to the base line Na Ba (Fig 2)

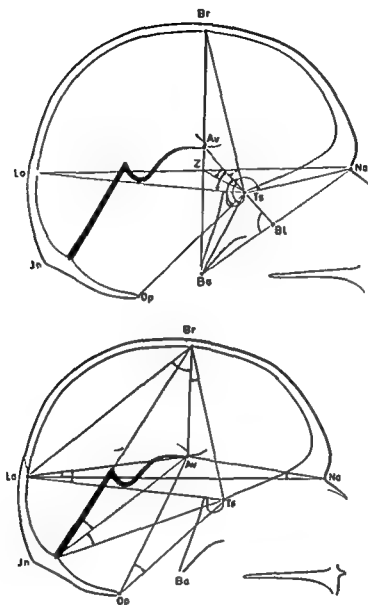


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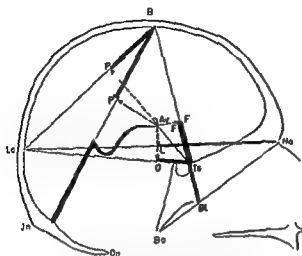


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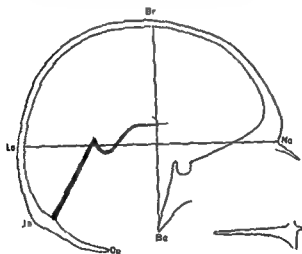


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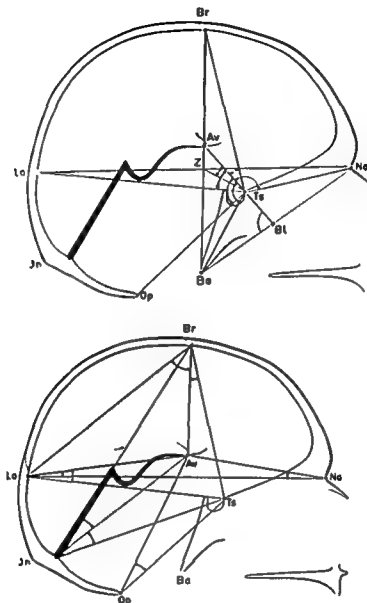


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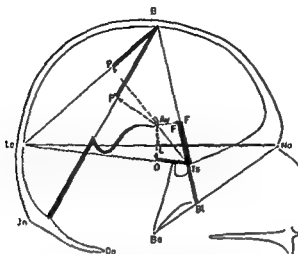


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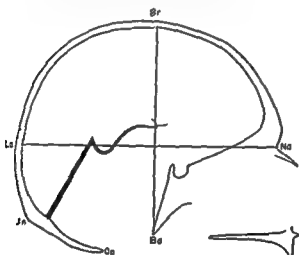


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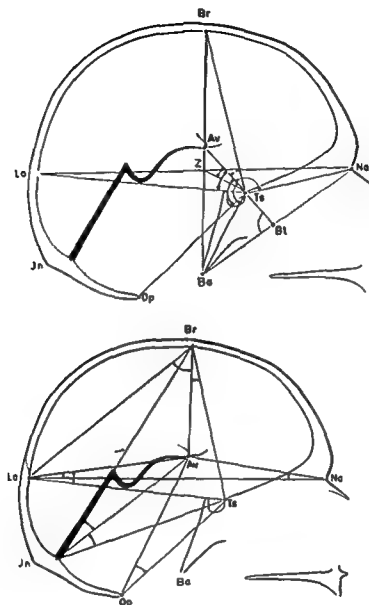


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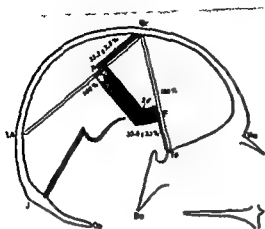


Fig 6 Exact method for determination of the localisation of the venous angle

3 On the bisection line of the angle tuberculum sellae endobregma (Fig 4)

4 On the point of intersection of the vertical lines erected on the anterior third of the line endobregma endolambda and on the inferior third of the line tuberculum sellae endobregma (Fig 5)

The exact method for determining the localisation of the venous angle is as follows (Fig 6)

The line between the tuberculum sellae and endobregma is divided in the ratio of 32.6 ± 3.1 to 67.4 and the line between endobregma endolambda is divided in the ratio of 33.2 ± 2.5 to 66.8 . The venous angle is situated on the intersection of the vertical lines erected at these points. The exact position of the venous angle can best be achieved from these two lines. The other lines as shown in Fig 2 as well as all angles will result in a larger zone of dispersion for the venous angle.

In analysing the reference system of the exact method it becomes evident that the line tuberculum sellae endobregma represents a datum for height while the line endobregma endolambda is a measurement for length. Both share the endobregma point. Height and length of the cranial vault are individually considered. Because of the connection of both reference lines in the endobregma point the alteration of size and shape of the cranial vault will be included. The essential change of shape of the cranial vault can be expressed by the altered inclination of the two reference lines towards each other. In addition the end points of the reference lines are chosen so that they lie in regions of lesser variability (HOWELLS 1957). If one compares this method with conventional processes of localisation as far as they are compatible

- MASPES P E ■ DONEGANI G: Flebografia cerebrale normale e pathologica. *Acta neurochir* 3 (1952) 147
- RING A: Variations in the sinuate and other cerebral veins affecting measurements of the venous angle. *Acta neurol* 52 (1959) 433
- UMBACH W: Untersuchungen zur Phlebographie der Hirngefasse. *Fortschr Röntgenstr* 77 (1952) 179
- WOLF B S, NEWMAN CH M and SCHLESINGER B: The diagnostic value of the deep cerebral veins in cerebral angiography. *Radiology* 64 (1955) 161

(MASPES & DONEGANI 1952, JOHANSON 1954, LIN et coll 1955, BRANDT 1959) our method improves the accuracy by 20—40 %

The deep cerebral veins gain more importance for the diagnosis of space occupying lesions by this exact method of localisation for the venous angle

SUMMARY

By taking into consideration the individual size and shape of the cranial vault the normal position of the venous angle of the deep cerebral veins can be localized approximately with the help of a few auxiliary lines and with exactitude by a new simple method

ZUSAMMENFASSUNG

Unter Berücksichtigung der individuellen Grösse und Form des Schädels lässt sich die normale Lage des Angulus venosus der tiefen Hirnvenen 1) durch einige Hilfslinien ungefähr und 2) durch eine neue einfache Methode genau bestimmen

RÉSUMÉ

On peut localiser approximativement l'angle veineux des veines cérébrales profondes en tenant compte des dimensions et de la forme de la voûte crânienne au moyen de quelques lignes auxiliaires. On peut le localiser exactement par une nouvelle méthode simple

REFERENCES

- BRANDT P. Über die Lage des Foramen Monroi im normalen und pathologischen seitlichen Phlebogramm und über die Verlagerung der tiefen inneren Venen bei pathologischen Prozessen des Grosshirnes. *Zbl Neurochir* 19 (1959) 130
- CURRY R. W. and CULBRETH G. G. The normal cerebral angiogram. *Amer J Roentgenol* 65 (1951) 345
- FISCHER E. Die Lagebesicherung der tiefen Hirnvenen zur Form des Schädels (mit Berücksichtigung der Verlagerung durch raumfordernde intracranielle Prozesse). Habilitationsschrift. Tübingen 1963
- HOWFELS W. W. The cranial vault: factors of size and shape. *Amer J Phys Anthropol* 15 (1957) 19
- JOHANSON C. The central veins and deep dural sinuses of the brain. *Acta radiol Suppl* 107 1954
- LAINE E., DELANDTSHEER J. M., GALIBERT P. et DELANTSHEER G. Étude phlébographique des tumeurs hémisphériques. *Neuro chirurgie* 1 (1955) 5
- LIN P. M., MOKROHNSKY J. F., STAUFFER H. M. and SCOTT M. The importance of the deep cerebral veins in cerebral angiography with special emphasis on the orientation of the foramen of Monro through the visualisation of the venous angle of the brain. *J Neurosurg* 12 (1955) 256

- MASPER P E & DONEGANI G. Flebografia cerebrale normale e pathologica. *Acta neurochir* 3 (1957) 147
- RING A. Variations in the striate and other cerebral veins affecting measurements of the venous angle. *Acta neurol* 52 (1959) 433
- UMBACH W. Untersuchungen zur Phlebographie der Hirngefäße. *Fortschr Röntgenstr* 77 (1952) 179
- WOLF B S, NEWMAN CH M and SCHLESINGER B. The diagnostic value of the deep cerebral veins in cerebral angiography. *Radiology* 64 (1955) 161

NEURORADIOLOGIC PROCEDURES IN CHILDREN

Comparison of heavy sedation and general anesthesia

by

R. V. GROOVER, A. M. CHUTORIAN and G. NELLHAUS

Since encephalography and cerebral angiography were first successfully performed on pediatric patients, many clinical and technical advances have been made. In recent years, attention has been given to methods of narcosis employed for these procedures (13). Until the beginning of this decade, general anesthesia was the most widely used. However, the widespread successful use of heavy sedation for cardiac catheterization in infants and children has encouraged its use in neurologic procedures. This report is a retrospective study of the comparative value of heavy sedation and general anesthesia in the performance of encephalography and cerebral angiography from a large pediatric neurology service.

Material. The report is based on 812 studies performed on 611 infants and children under 13 years of age admitted to the Babies Hospital and the Neurological Institute of New York between January 1955 and June 1964. The criteria used here are identical to those used in an earlier report (13). A study constitutes each time a patient was either anesthetized or sedated, regardless of the number of procedures performed. Patients who underwent surgery

Table 1
Age distribution

Age in years	Encephalography		Angiography		Combined examinations	
	Anesthesia	Sedation	Anesthesia	Sedation	Anesthesia	Sedation
< 1	41	150	8	24	2	5
1-4	61	84	27	39	3	7
5-8	59	68	23	35	2	7
9-12	31	74	20	55	3	8
Total	192	376	74	153	10	27

within 24 hours of their examination are excluded because of the difficulty in distinguishing between complications due to the diagnostic procedures and those due to surgery. The patients in both groups were similar with respect to age distribution (Table 1) as well as clinical status and indications for study. A total of 276 studies were performed under general anesthesia and 536 under heavy sedation. These represent 548 encephalographies, 227 cerebral angiographies, and 37 combined examinations. Of the 644 patients 519 were examined once, 99 twice, 12 three times, 12 four times, and one each five and six times.

Techniques. All patients were examined upon admission to the hospital and prior to roentgenologic procedures. Every attempt was made to have the patient in the best possible condition for the procedures. Approximately 75 % of the studies were performed by members of the pediatric neurology service, and 25 % by members of the neurosurgery or neuroradiology departments. All were performed under the supervision of the Department of Neuroradiology, thus affording considerable standardization in technique despite the number of individuals involved.

Encephalography was performed with air by the limited fractional replacement method (11, 20). The lumbar route was used in the majority of instances, but in small infants this was sometimes combined with direct ventricular puncture via the anterior fontanel.

Angiography was performed by the percutaneous approach in all but 2 cases using a No. 18 or 20 Courmand needle. The contrast material used since 1959 has been methylglucamine diatrizoate (Renografin). Previously Iodopyracet (Diodrast) and sodium diatrizoate (Hypaque) were used in some patients. The amount injected varied with the size of the patient, the abnormality

Table 2
Sedative dosages for narcosis in children

Age in years	Secobarbital mg/kg	Chlorpromazine mg/kg	Meperidine mg/kg	Atropine
< 1-4	8-10	1.5	1-1.5	0.05 mg/kg
5-8	5-7	1.0	1-1.5	0.3 mg/pt
9-12	3-4	0.75	1.0	0.4 mg/pt

suspected, and the number of injections required. Prior to arterial injections, patients were given skin and/or intravenous sensitivity tests. Since 1957 a mechanical injector has been used. Previously, manual injections were employed. Simultaneous biphasic interoposterior and lateral serial films were made with each injection. General anesthesia, the most frequently employed method of narcosis from 1955 to 1960, was administered by supervised anesthesiology residents. Premedication drugs varied and were ordered by the anesthesiologist. A nitrous oxide-oxygen-ether mixture and thiopental (Pentothal) were the anesthetics employed most frequently. Intubation was usually, but not always, employed.

After 1960 heavy sedation was used most frequently for narcosis. The sedative mixture used included secobarbital, chlorpromazine, meperidine and atropine (Table 2). It should be emphasized that the dosage table served as a general guide and was altered according to the procedure and the condition of the patient. Generally, moderate doses of secobarbital plus moderate amounts of meperidine were given for arteriography, while the higher dosages of secobarbital and little or no meperidine were used in encephalography. Chlorpromazine was generally used in both procedures, except in cases of suspected brain stem or hypothalamic lesions, where it was used with great caution. Atropine was generally employed in all procedures. The appropriate mixture was given intramuscularly 45 to 60 minutes prior to the procedure, and was supplemented by local anesthesia of the puncture site at the time of the procedure. Occasionally, additional secobarbital or meperidine was required during the procedure if sedation was inadequate. This was given either intravenously or intramuscularly as the situation indicated.

Results

The degree of technical success was similar in both groups (Table 3). In the anesthetized group, 6.2% of the examinations were incomplete and inadequate for diagnostic purposes, as compared to 5.6% of the sedated group. The main

Table 3
Results of the neuroradiologic examinations

Procedure and narcosis	Number of examinations	Unsatisfactory/incomplete	
		Number	Percentage
<i>Anesthesia</i>			
Encephalography	192	11	5.7
Angiography	74	6	8.1
Combined examinations	III	—	—
Total	276	17	6.2
<i>Sedation</i>			
Encephalography	356	23	6.5
Angiography	153	7	4.5
Combined examinations	27	—	—
Total	536	30	5.6

reasons for failures were the introduction of subdural air or difficulty in cannulating the artery. Untoward reactions to the procedure were a less frequent cause. Such reactions occurred during 9 encephalographies, 3 under anesthesia and 6 under sedation.

Regardless of the type of narcosis used, most patients required 3 to 6 hours and occasionally 8 or more hours to become fully alert. In the sedated group, the majority of patients were taking and retaining fluids within 2 to 3 hours after their study, whereas in the anesthetized group post-anesthesia nausea and vomiting delayed oral intake longer. Generally patients who received inhalation anesthesia were routinely placed in croupettes for 24 hours after their procedures. Such was seldom required in patients in the sedated group. Supplementary intravenous fluids were required more often in the anesthetized group. Following encephalography, approximately 50% of patients in both groups exhibited fever and/or signs of meningeal irritation lasting 24 to 48 hours. It was not unusual for temperatures to reach 39.4° C per rectum during this time. However, the incidence of temperature elevations above 39° C per rectum dropped sharply after 48 hours.

In 106 (13%) of the 812 studies, one or more complications were encountered which were in excess of the expected reactions. A total of 64 (7.9%) occurred in the anesthetized group while 42 (5.1%) occurred in the sedated group. For purposes of classification, these complications are divided into 3 types (Table 4):

1. Significant fever and/or meningeal reaction
2. Local and/or miscellaneous, Significant fever and/or meningeal reactions designates those patients who
3. Respiratory and/or cardiovascular

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Age in years	Secobarbital mg/kg	Chlorpromazine mg/kg	Meperidine mg/kg	Atropine
< 1-4	8-10	1.5	1-1.5	0.02 mg/kg
5-8	5-7	1.0	1-1.5	0.3 mg/pt
9-12	3-4	0.75	1.0	0.4 mg/pt

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Results

The degree of technical success was similar in both groups (Table 3). In the anesthetized group, 6.2% of the examinations were incomplete and inadequate for diagnostic purposes, as compared to 5.6% of the sedated group. The main

means while the other required suturing of a rent in the carotid artery. Allergic reactions to contrast material occurred in 3 instances. Neurologic sequelae were rare and generally transient. One child with a clinical diagnosis of degenerative central nervous system disease was studied under general anesthesia. He experienced significant hypoxia during the procedure and had his first seizure 3 days after the encephalography. Three sedated patients exhibited transient hemiparesis following the procedure — two encephalographies and one combined examination. A child with a brain stem glioma exhibited signs of block and increased intracranial pressure following encephalography requiring ventricular drainage for relief.

The respiratory and/or cardiovascular complications are subtyped into fatal, serious, and less serious. Serious designates notable hypoxia such as laryngospasm, apnea, cardiac arrest, prolonged arrhythmia and the like. Less serious designates brief bronchospasm, hypotension, tachycardia and croupiness. Such complications were encountered in 20 % of the patients studied under general anesthesia and in 4.7 % of the patients studied under heavy sedation. All 3 fatalities occurred in patients examined under general anesthesia (Table 4).

Less serious complications were encountered in 51 instances — 36 in the anesthetized group and 15 in the sedated group. A total of 27 patients of the former group had to be kept in oxygen and mist for 36 hours or more because of laryngo-tracheitis, bronchitis and/or pneumonia. Two exhibited notable transient respiratory distress during induction. In 7 infants of less than 3 years transient hypothermia and hypotension was observed following encephalography. Of the patients belonging to the sedated group 11 exhibited bronchitis or bronchopneumonia, 11 exhibited transient respiratory distress either during or after the procedure, and 2 exhibited transient mild cardiac arrhythmias.

In all 26 serious complications were encountered — 16 in the anesthetized group and 10 in the sedated group. In the former group apnea or respiratory obstruction ensued necessitating active respiratory assistance. In 7 patients severe laryngeal edema, pneumonia or other serious respiratory difficulties were encountered. In 7 sedated patients severe apnea and/or cardiac arrest occurred requiring intubation, assisted respirations, and in 2 instances external cardiac massage. Irregular respirations, labile blood pressures and cardiac arrhythmias were encountered in 2 patients with brain stem glioma following encephalography, and severe circulatory collapse occurred during an arteriography under heavy sedation in a child with a craniopharyngioma.

Three deaths were encountered in the total of 813 studies. All three occurred in patients given general anesthesia, i.e. an overall mortality rate of 0.37 % or a rate of 1.1 % of procedures performed under general anesthesia.

Table 4
Complications in children

Procedure and anarcosis	No of examina- tions	With compli- cations		I ever and/or meningeal		I ocal and mis- cellaneous		Respiratory and/or cardiovascular					
								Fatal		Serious		Less serious	
		No	%	No	%	No	%	No	%	No	%	No	%
<i>Inesthesia</i>													
Encephalo- graphy	192	38	19.8	6	3.1	2	1.0	1	0.5	11	5.7	21	10.9
Angiography	74	22	29.7	1	1.4	7	9.5	2	2.7	3	4.1	14	19.0
Combined	10	4	40.0	0	0	1	10.0	0	0	2	20.0	1	10.0
Total	276	64	23.2	7	2.5	10	3.6	3	1.1	16	5.8	36	13.0
<i>Sedation</i>													
Encephalo- graphy	336	21	6.7	8	2.2	3	0.8	0	0	11	2.2	10	2.7
Angiography	133	15	9.9	1	0.7	7	4.5	0	0	2	1.3	5	3.3
Combined	27	3	11.1	3	11.1	0	0	0	0	0	0	0	0
Total	36	12	7.8	12	2.2	10	1.9	0	0	10	1.9	15	2.8

exhibited temperatures greater than 39° C per rectum and/or significant meningeal reactions lasting more than 48 hours. Such complications were similarly encountered in both groups (Table 4). There was one case of bacterial meningitis following encephalography. This occurred in a hydrocephalic infant with a previous Spitz-Holter shunt and two prior, ostensibly eradicated, episodes of staphylococcus albus bacteremia. Three cases of severe aseptic meningitis occurred following encephalographies performed on successive days by different physicians. All cultures were negative and it was presumed that probably some chemical irritant had been present on the equipment used. In the remaining 13 instances, the fever and meningeal signs subsided with supportive therapy and no cause other than the procedure could be implicated. In the two incidences of fever following angiography, one occurred in a child with pseudotumor cerebri and the second in an infant with infantile hemiplegia.

Local and miscellaneous complications include excessive hematoma formation, allergic reactions to contrast material and neurologic sequelae. Hematomas of such size as to produce tracheal shift, prolonged hoarseness and/or difficulties in swallowing occurred in 8 instances following angiography — 5 in the anesthetized group and 3 in the sedated group. In 3 instances persistent bleeding from the puncture site was encountered. Two were controlled by local

means, while the other required suturing of a rent in the carotid artery. Allergic reactions to contrast material occurred in 3 instances. Neurologic sequelae were rare and generally transient. One child with a clinical diagnosis of degenerative central nervous system disease was studied under general anesthesia. He experienced significant hypoxia during the procedure and had his first seizure 3 days after the encephalography. Three sedated patients exhibited transient hemiparesis following the procedure — two encephalographies and one combined examination. A child with a brain stem glioma exhibited signs of block and increased intracranial pressure following encephalography, requiring ventricular drainage for relief.

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Complications in children

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			No	%	No	%	No	%	Total		Serious		Less serious	
<i>Anesthesia</i>														
Encephalo- graphy	192	38	19.8	6	3.1	2	1.0	1	0.5	11	5.7	21	10.9	
Angiography	74	22	29.7	1	1.4	7	9.5	2	2.7	3	4.1	14	19.0	
Combined	10	4	40.0	0	0	1	10.0	0	0	2	20.0	1	10.0	
Total	276	64	23.2	7	2.5	10	3.6	3	1.1	16	5.8	36	13.0	
<i>Sedation</i>														
Encephalo- graphy	356	24	6.7	8	2.2	3	0.8	0	0	8	2.3	10	2.7	
Angiography	153	15	9.9	1	0.7	7	4.5	0	0	2	1.3	5	3.3	
Combined	27	3	11.1	3	11.1	0	0	0	0	0	0	0	0	
Total	536	42	7.8	12	2.2	10	1.9	0	0	10	1.9	15	2.8	

exhibited temperatures greater than 39° C per rectum and/or significant meningeal reactions lasting more than 18 hours. Such complications were similarly encountered in both groups (Table 4). There was one case of bacterial meningitis following encephalography. This occurred in a hydrocephalic infant with a previous Spitz-Holter shunt and two prior, ostensibly eradicated, episodes of staphylococcus albus bacteremia. Three cases of severe aseptic meningitis occurred following encephalographies performed on successive days by different physicians. All cultures were negative and it was presumed that probably some chemical irritant had been present on the equipment used. In the remaining 13 instances, the fever and meningeal signs subsided with supportive therapy and no cause other than the procedure could be implicated. In the two incidences of fever following angiography, one occurred in a child with pseudotumor cerebri and the second in an infant with infantile hemiplegia.

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Three deaths were encountered in the total of 813 studies. All three occurred in patients given general anesthesia, i.e. an overall mortality rate of 0.37 % or a rate of 1.1 % of procedures performed under general anesthesia.

Table 5

Age of patients and incidence of complications

Narcosis	No of examinations	Complications	Percentages
<i>Anesthesia</i>			
< 1 year	51	20	39
1—4 years	86	23	27
5—8 years	85	16	19
9—12 years	51	5	9
Total	276	64	23.2
<i>Sedation</i>			
< 1 year	159	15	9.4
1—4 years	130	15	11.6
5—8 years	110	6	5.6
9—12 years	137	6	4.4
Total	536	42	7.8

Case reports

Case 1 The first fatality was a 3 month old female weighing 4.5 kg with bilateral subcapsular cataracts, microcephaly and diarrhea of unknown etiology. She was initially premedicated with 550 mg tribromethanol rectally which was partially expelled. An additional 250 mg tribromethanol was given rectally and 20 mg pentobarbital was given intramuscularly. General inhalation anesthesia was then administered without intubation. Following the removal of 3.0 ml of spinal fluid and the instillation of 10 ml air via the lumbar subarachnoid space respiratory and cardiac arrest ensued. The patient was promptly intubated and open cardiac massage instituted but to no avail.

Case 2 The second death occurred in an 8 week old female weighing 5 kg with a massive hemangioma of the left face and scalp. Intracranial extension was ruled out by a left common carotid arteriogram performed under general anesthesia. Ligation of the left external carotid was planned but the patient experienced cardiac arrest while being transferred from angiography to the operating room. Open chest cardiac massage was performed with return of cardiac action after 3 minutes. The infant never regained consciousness and expired 5 days later.

Case 3 The third death occurred in an 8 year old female weighing 30 kg who had an acute severe intracerebral and subarachnoid hemorrhage secondary to a ruptured arteriovenous malformation of the right frontal lobe. She was stuporous but arousable before the examination and was given general anesthesia and intubated. A left common carotid angiography with a cross compression test was performed followed by left vertebral angiography. During the procedure the patient exhibited signs of tonsillar and tentorial herniation which were treated with intravenous urea. Subsequently she developed respiratory arrest and frank pulmonary edema requiring assisted respirations for the 12 following hours. She never regained consciousness and expired 3 days later.

In both groups proportionately greater incidence of complications was encountered in patients under 5 years of age. This incidence was significantly greater in patients studied under general anesthesia. This was most marked in the infants under 1 year where the complication rate was four times greater in patients studied under general anesthesia (Table 5).

Discussion

The occurrence of three fatalities in the anesthetized group might be considered merely fortuitous; however, one cannot escape the fact that no deaths occurred in almost twice as many procedures performed on comparable patients under heavy sedation. The two deaths occurring in infants under 1 year of age were felt by the physicians in charge to be due primarily to the effects of anesthesia and their general conditions rather than to the procedures performed. Careful review of the charts substantiated this. The third death was most likely due to the severity of the child's illness and to the complications occurring during the study rather than to the effects of general anesthesia per se.

Significant and/or meningeal reactions and local and/or miscellaneous complications are attributable to the procedures themselves. The incidence of such complications was similar in both the anesthetized and sedated groups. However, notable difference in the incidence of respiratory and/or cardiovascular complications was apparent between the two groups; for such complications occurred approximately 4.5 times more frequently in examinations performed under general anesthesia (Table 4). Since other factors were comparable, it may be assumed that the primary reason for the significant difference was in the method of narcosis used. Support for such a conclusion may be found by reviewing the inherent risks of general anesthesia in pediatric patients (1, 15, 19).

General anesthesia has generally been advocated for narcosis in neuroradiologic procedures in pediatric patients for two reasons — to decrease the incidence of technical failures and to decrease the incidence of local complications (3, 4, 8, 10). However, in our experience, 6.2% of the studies performed under general anesthesia were incomplete and unsatisfactory, while 5.6% of the studies performed under heavy sedation were unsatisfactory. Local complications such as excessive hematoma formation, injury to the arterial wall, and the like occurred in 3.6% of the procedures performed under general anesthesia and in 1.9% of studies under heavy sedation.

In addition to the obvious advantage of reducing the incidence of cardiorespiratory complications, the use of heavy sedation obviates the need for the direct attendance of an anesthesiologist. Technically, the procedures are more

easily performed in the absence of elaborate anesthetic equipment. The time required for the performance is reduced, since the patient arrives in the radiology department in the sedated state. Following the roentgenologic examination the patient is less prone to nausea and vomiting, takes oral fluids earlier, and requires less nursing care in the post study period than the comparable patient who has had general anesthesia. Thus, heavy sedation has been the narcosis of choice for the performance of encephalography and cerebral angiography in pediatric patients in our department since 1960. General anesthesia has been reserved for those patients who are to be operated upon immediately after the examination.

The use of sedation for narcosis in neuroradiologic procedures in infants and children is not new. A detailed review of the subject by two of the authors is set forth in an earlier report (13). In 1930, CROTHERS and his coworkers suggested the use of repeated small doses of morphine in encephalography. Four years later, RUPILIUS advocated sedation for encephalography, pointing out the dangers of general anesthesia. More recently BAMBURGER & MATTHES suggested a combination of chlorpromazine, promethazine and meperidine for patients undergoing encephalography.

The precise amount and type of sedation an infant or child may require in order that a given procedure may be successfully performed remains largely a matter of experience. This involves many variables, such as the clinical status of the patient, the skill and speed of the operator and roentgen technician, and the extent and duration of the examination. The sedative mixture used in this report included secobarbital, chlorpromazine, meperidine and atropine (Table 2). It should be emphasized that this dosage schedule was not absolute, but rather served as a guide and was varied according to the procedure and the condition of the patient. Also, this combination of drugs is not to be considered the best or the only satisfactory sedative mixture, for there appears to be no universal agreement as to which drug or combination of drugs is best (7, 14). Rather it may be said that this combination of drugs has been successful in our hands. It is interesting to note that combinations of drugs similar both quantitatively and qualitatively to the drugs outlined here have been recommended as preanesthetic medication for infants and children (4, 9, 17, 18, 19).

Little or no meperidine in combination with the outlined dosages of secobarbital, chlorpromazine and atropine was given for encephalography because of the tendency for meperidine to produce hypotension and emesis. These appear to be exaggerated by the postural gyrations in encephalography. The patients sedated for arteriography usually received the lower dosage of secobarbital, in addition to meperidine, chlorpromazine, and atropine, because of the pain associated with cannulation of the artery. Chlorpromazine was

used in very low dosages if at all in patients with suspected hypothalamic or brain stem lesions because of the distinct impression that such patients were especially sensitive to the drug. Atropine was used in both types of procedures primarily because of its inhibition of reflex vagal activity and depression of pressoreceptors.

The main reasons for inadequate sedation of patients in the present series was either miscalculation on the part of the operator as to the amount of sedation needed or unavoidable delays in starting the procedure so that the optimal period of sedation had passed. Adequate sedation in such cases was usually achieved by giving a supplemental injection of either secobarbital or meperidine intravenously or intramuscularly as the situation indicated. Oversedation was an extremely rare problem. In one instance a small infant received twice the ordered amount of sedation. His procedure was cancelled and performed successfully under more reasonable sedation at a later date. He tolerated the seemingly alarming overdosage very well. The importance of administering the sedative mixture at least 45 to 60 minutes before the procedure cannot be overemphasized. If the patient is unduly disturbed before maximal sedative effect has taken place, we have found the patient reacts suboptimally to what would otherwise have been adequate sedation. However, if the medication is given 45 to 60 minutes before the procedure, we have found the patient arrives in the radiologic department sedated and sleeping. Generally he arouses somewhat when positioned but promptly returns to sleep when left alone. He usually arouses again when the area of the puncture site is infiltrated with local anesthesia. However, if then left undisturbed for a few minutes, he returns to his ataractic state and the operator may proceed with the examination.

Conclusions

A total of 812 neuroradiologic examinations, not followed in 24 hours by surgery in infants and children under 13 years of age, have been reviewed retrospectively. Of these, 276 were performed under general anesthesia while 536 were performed under heavy sedation. The two groups were similar with respect to age distribution and clinical status of the patients, indications for roentgen examination, and methods used. The methods of narcosis employed in the patients were outlined and discussed.

Complications were encountered in 106 (13%) of the 812 procedures. Sixty-four (23.2%) of the examinations performed under general anesthesia and 42 (7.8%) of those performed under heavy sedation were complicated. Complications attributable to the procedures themselves were essentially comparable in both groups, whereas respiratory and cardiovascular complica-

tions were encountered 4-5 times more frequently in the anesthetized patients. This preponderance was evident in all age groups, but it was most marked in infants under one year. The degree of technical success under both types of narcosis was comparable. Thus, it has been shown that neuroradiologic procedures in pediatric patients can be performed under heavy sedation with promise of comparable technical success and with less risk to the patient than under general anesthesia.

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ZUSAMMENFASSUNG

Es wurden insgesamt 812 neuroradiologische Studien von Kleinkindern und Kindern unter 13 Jahren retrospektiv durchgesehen. Ein chirurgischer Eingriff innerhalb 24 Stunden nach der neuroradiologischen Untersuchung war nicht vorgenommen worden. In 276 Fällen war die Untersuchung in allgemeiner Anästhesie, in 536 Fällen nach Verabreichung von starken Sedativa durchgeführt worden. Beide Gruppen sind sich ähnlich in der Altersverteilung, im klinischen Status, den Indikationen und Untersuchungsmethoden. Die angewandten Methoden der Narkose werden aufgezeigt und besprochen.

RÉSUMÉ

Les auteurs ont passé en revue 812 examens neuroradiologiques chez des nourrissons et des enfants de moins de 13 ans qui n'ont pas été suivis d'intervention chirurgicale dans les 24 heures. 276 de ces examens avaient été faits sous anesthésie générale et 536 sous sédation profonde. Les deux groupes étaient semblables en ce qui concerne la distribution selon l'âge, l'état clinique des malades, les indications et les méthodes utilisées. Les auteurs décrivent et étudient les méthodes de narcose employées.

REFERENCES

- 1 ADRIANI J Anesthesia in infancy and childhood G P 27 (1963) 82
- 2 BAMBERGER P and MATTHIES A Anfalle im Kindesalter p 277 S Karger Basel 1959
- 3 BRANDT S GARDP M de LA and ROSENDAL T The technique of pneumoencephalography in children Acta paediat Suppl 103 (1955) 61
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- 7 DOLCHY A G The evaluation of premedication in children Proc roy Soc Med 52 (1959) 873
- 8 FARMER T W Ed Pediatric Neurology Hoeber Medical Division Harper & Row New York Evanston and London 1964
- 9 FREEMAN A and BACHMANN L Pediatric anesthesia An evaluation of preoperative medication Anesth Analg 38 (1959) 429
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- 13 NEELIHALS G and CHUDOMIRAN A Narcosis for neuroradiologic procedures in children Arch Neurol 10 (1964) 485
- 14 RACKOW H and SALANTER E A dose effect study of preoperative medication in children Anesthesiology 23 (1962) 747
- 15 — — and GREEN L T Frequency of cardiac arrest associated with anesthesia in infants and children Pediatrics 28 (1961) 697
- 16 RUPILUS K Über Zerebrale Störungen im Kindesalter und ihre Enzephalographische Diagnostik Arch Kinderheilk 102 (1934) 194 103 (1934) 39
- 17 SANDOZ M S and FRYE T J Preoperative sedation and production of a quiescent state in children promethazine mependone scopolamine sedation J Amer med Ass 164 (1957) 1729
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THE THALAMOPERFORATE ARTERY

by

KAZUO HARA and YASUSADA FUJINO

The small arteries arising from the posterior part of the circle of Willis have received little attention in the literature. The increased use of vertebral angiography has however led to demonstration and the technique evolved at our centre has proved valuable in the diagnosis of deep seated cerebral tumours.

Autopsy studies

Twenty two fresh brain specimens were used in an effort to form estimates of the normal anatomic patterns of the small arteries emerging from the posterior part of the circle of Willis. The basilar arteries having been injected with gelatin bromium solution (LINDBLOM, JOHANSON), the brains were fixed in 10 % formalin solution. The macroscopic and roentgenographic findings were then reviewed.

The many small arteries that arise from the posterior part of the circle of Willis may be divided into the superficial branches, which reach the surface of the brain, and the perforating branches, which pass through the cerebral parenchyma. The latter are mostly more than 0.2 mm in diameter and are outlined in the vertebral angiogram, while the superficial branches, less than 0.2 mm in diameter, are difficult or impossible to show (Fig. 1).



Fig 1 Autopsy specimen Thalamoperforate artery (→) and posterior communicating artery (→)



Fig 2 Sagittal section through normal autopsy specimen showing external part of thalamoperforate artery (arrow)



Fig 3 Lateral roentgenogram of normal autopsy specimen Thalamoperforate artery (arrow) and transition between external part and parenchymal part (needle point)

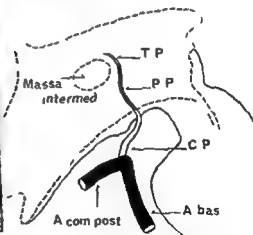


Fig 4 Diagram of division of the thalamoperforate artery CP Cisternal part PP Parenchymal part TP Terminal part

Branches emerging from the terminal part of the basilar artery Some perforating branches arise from the terminal part of the basilar artery. These may range in number from two to six and in size from 0.2 to 0.8 mm and arise proximal to the bifurcation of the basilar artery and the posterior communicating artery.

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Fig 1 Autopsy specimen Thalamoperforate artery (→) and posterior communicating artery (---)



Fig 2 Sagittal section through normal autopsy specimen showing cisternal part of thalamoperforate artery (arrow)



Fig 3 Lateral roentgenogram of normal autopsy specimen Thalamoperforate artery (arrow) and transitional region between cisternal part and parenchymal part (needle point)

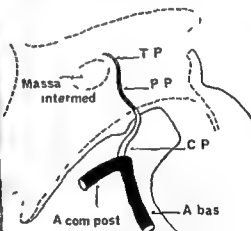


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Fig 6 Variations of external part of thalamoperforate artery (solid line)



Fig 7 Variations of parenchymal part of thalamoperforate artery (solid line)

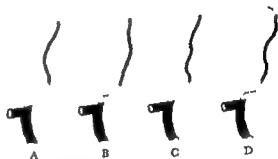


Fig 8 Lateral roentgenogram of autopsy specimen. The perforating branch arises from the posterior communicating artery (arrow).

main types, A, B, C and D, the characteristic variations being well displayed in the lateral view (Fig 6). Most of the 22 cases of the present series were of Type A, but some examples of a combination of two or more types were also recognized.

The thalamoperforate artery may also be divided into Types A, B, C and D in its parenchymal part (Fig 7). This part of the artery runs upwards in one or two curves with a tendency for the right and left sides to be symmetrical.



Fig. 5. A p. roentgenogram of normal autopsy brain. 1. parenchymal part (→) and terminal part (---→)

One of these arteries was termed the thalamoperforate artery by reason of its relation to the thalamus by HAYMAKER or *artère du pédicule thalamoperforé* by TESTUT. The thalamoperforate artery usually runs backwards through the interpeduncular fossa where it gives off one or two ramuli. As it leaves the basilar artery, it follows the sigmoid curve to reach the parenchyma where it runs vertically into the perforated substance (Fig. 2). The remainder of the branches arising from the more distal part of the terminal part of the basilar artery and some branches of the thalamoperforate artery are distributed over the cerebral peduncles.

Branches emerging from the posterior communicating artery. These may also be divided into perforating and superficial branches. The latter are very small and usually penetrate the lateral perforate area so named by KOMATSU.

Roentgenography

A study of sagittal section of the cerebral hemispheres at the level of the circle of Willis (Fig. 3) indicates that the thalamoperforate artery may be divided into three parts: cisternal, parenchymal and terminal (Fig. 4). The cisternal part of the thalamoperforate artery lies in the extracerebral space from which it runs upwards and slightly forwards along the wall of the third ventricle through the parenchyma. The parenchymal part courses outwards and forwards or outwards and backwards to become the terminal part after passing the third ventricle. Filling of the thalamoperforate artery and its relation to surrounding structures are well shown in an a.p. roentgenogram of an autopsy specimen (Fig. 5). The terminal part of the thalamoperforate artery is seen in the lateral view to pass round the intermediate mass, however, the terminal part is not always clearly demonstrated.

The thalamoperforate artery in its cisternal part may be divided into four



Fig 13 Stenosis of Sylvian duct producing dilatation of third ventricle and stretching of parenchymal part of thalamoperforate artery (arrow)



Fig 14 Stretching and forward and downward displacement of parenchymal part of thalamoperforate artery in pinealoma (arrow)



Fig 15 Tumour arising from the quadrigeminal plate stretching and forward displacement of thalamoperforate artery (arrow)



Fig 16 Infiltrating tumour in thalamus irregularity of thalamoperforate artery (arrow) and pathological vessels

suitable for study and proved to be particularly informative (Fig 9). Minor movements of the head or poor contrast filling may however produce blurring of the artery. The transitional portion between its cisternal and parenchymal parts lies in a straight line as seen at autopsy, an appearance characteristic enough to allow easy differentiation of the two parts.

The branches arising from the posterior communicating artery visible in a vertebral angiogram in a quarter of 60 cases are commonly observed in young



Fig 9 Vertebral angiogram. Normal thalamoperforate artery (arrow)



Fig 10 Filling of entire perforating branch from posterior communicating artery (→) and thalamoperforate artery (↔)



Fig 11 Normal vertebral angiogram with bilateral carotid compression showing perforating branch from posterior communicating artery (arrow) and thalamoperforate artery



Fig 12 Stretching and backward displacement of thalamoperforate artery in cranio-pharyngioma

Type A was frequently observed and Types C and D with two gentle curves were noted in a few cases. Some branches occasionally reached the hypothalamic region. The perforating branches arising from the posterior communicating artery may be divided into cisternal and parenchymal, although classification is impossible because of unusual variations in the course of these branches (Fig 8).

As the thalamoperforate artery is usually superimposed on the cerebellar or other arteries in the ap vertebral angiogram, only the lateral view was



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Fig 10 Filling of entire perforating branch from posterior communicating artery (x) and thalamoperforate artery (x-x)



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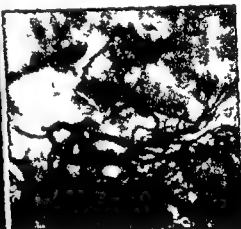


Fig. 16 Infiltrating tumour in thalamus. Irregularity of thalamoperforate artery (arrow) and pathologic vessels

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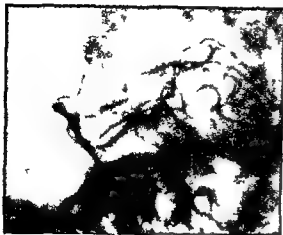


Fig. 17 Backward displacement of basilar artery in clival tumour

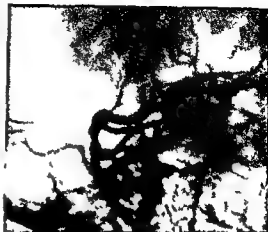


Fig. 18 Hemangioblastoma of cerebellum. Stretching of superior cerebellar artery, signs of angioma and slight stretching and upward and forward displacement of all parts of thalamo-perforate artery



Fig. 19 Cerebellopontine angle tumour. Upward displacement of superior cerebellar artery, posterior cerebral artery and cisternal part of thalamoperforate artery

subjects (Fig. 10). Filling of these arteries is readily obtained by bilateral carotid compression during angiography (Fig. 11).

Influence of tumours on the thalamoperforate artery. The thalamoperforate artery is displaced and deformed in most cases of space-occupying lesions arising in the deep cerebral region around the thalamus and midbrain or in the posterior fossa. Fig. 12 shows that the artery is stretched and displaced backwards by a

craniopharyngioma which is distended posteriorly. The parenchymal part of the artery is seen to be stretched but no change in the cisternal part is evident in a case of dilatation of the third ventricle, these appearances were believed to be due to the dilatation of the third ventricle caused by stenosis of the Sylvian duct (Fig 13). Stretching and displacement forwards and downwards of the thalamoperforate artery are also evident in its parenchymal part with no changes in its cisternal part in a case of pinealoma in Fig 14. Stretching and forward displacement of the thalamoperforate artery occurred in a case of a tumour arising in the quadrigeminal plate (Fig 15). A tumour infiltrating the thalamus has caused irregularity of the thalamoperforate artery in Fig 16. Backward displacement of the thalamoperforate artery without stretching and deformity is evident in a case of a clival tumour in which the basilar artery is also displaced to the rear (Fig 17). In a case with a cerebellar tumour the thalamoperforate artery is displaced slightly upwards and forwards in all its parts but stretching is not marked (Fig 18). Fourteen out of 17 cases of cerebellar tumour were found to present these changes. Slight upward displacement of the cisternal part of the thalamoperforate artery with slight stretching of its parenchymal part was present in a case of a cerebellopontine angle tumour (Fig 19). These findings were observed in 9 out of 10 cases of this condition.

The importance of performing vertebral angiography in the diagnosis of deep seated lesions thus becomes apparent. It would appear that arteriography of the thalamoperforate artery provides additional useful information in the study of intracranial lesions and adds a great deal to the knowledge of the vascular patterns that may be encountered in various conditions.

Acknowledgement

This study was supported by a Research Grant from the Waksman Foundation of Japan.

SUMMARY

A review is presented of the roentgen anatomical correlations of the vascular patterns of the small arteries particularly the thalamoperforate artery arising from the posterior part of the circle of Willis and effects caused by tumours.

ZUSAMMENFASSUNG

Es wird eine Übersicht über die Röntgenanatomie der kleinen Arterien gegeben. Im besonderen wird über die Art thalamoperforata, die vom rückwärtigen Abschnitt des Circulus Willisii entspringt und ihre Beeinflussung durch Tumore berichtet.

RÉSUMÉ

Étude des corrélations radio anatomiques des petites artères issues de la partie postérieure du cercle de Willis et en particulier l'artère thalamo perforée et des modifications causées par les tumeurs

REFERENCES

- ANDERSON P T The lenticulo striate arteries and their diagnostic value A preliminary report *Acta radiol* 50 (1958), 84
- BEVOR C E The cerebral arterial supply *Brain* 30 (1907) 403
- ECHEA A The normal cerebral angiogram Charles C Thomas Springfield Ill 1951
- GALLOWAY J R and GREITZ T The medial and lateral choroid arteries An anatomic and roentgenographic study *Acta radiol* 53 (1960) 353
- HAYMAKER W Handbuch der speziellen pathologischen Anatomie und Histologie (Henke-Lubarsch) Springer Verlag, Berlin Göttingen Heidelberg 1957
- HUBNER A Zur Topographie der Ernährungsgebiete der einzelnen Hirnarterien *Zbl med Wiss* 52 (1872) 817
- JOHANSON C The central veins and deep dural sinuses of the brain An anatomical and angiographic study *Acta radiol Suppl* (1954) 107
- KODAMA Y Atlas of human anatomy Vol V Kanehara-shuppan Tokyo Japan 1956
- KRAVITZ H and RICHTER H R Die cerebrale Angiographie Georg Thieme Verlag Stuttgart 1952
- LINDBLOM K A roentgenographic study of the vascular channels of the skull *Acta radiol Suppl* (1936) 30
- LINDNERO R Handbuch der speziellen pathologischen Anatomie und Histologie (Henke-Lubarsch) Springer Verlag Berlin Göttingen Heidelberg 1957
- SJOGREN S T The anterior choroidal artery *Acta radiol* 46 (1956) 143
- SUZUKI M Studies on the distribution of the basal branches of human cerebral arteries (Japanese) The Sapporo Medical Journal 19 (1961) 307
- TESTUT L Traité d'anatomie humaine Gaston Doin & Cie Paris 1929
- WESINER G The recurrent artery of Heubner and the arteries of the central ganglia *Acta radiol Diagnosis* (1963) 919

RADIOLOGICAL INVESTIGATION OF GLOMUS JUGULARE TUMOURS

by

T. DESMOND HAWKINS

The clinical and radiological features of glomus jugulare tumours have been widely recognised and reported since the original suggestion by ROSENWASSER in 1915 that his case of a carotid body tumour of the middle ear and mastoid might have originated in the glomus jugulare.

A review of 316 case histories in the English literature showed that in 42 % there was no report of a roentgen examination (ALFORD & GUILFORD 1962). Even in some of the more recently reported cases the part that radiology can play in the investigations of these tumours has not been fully exploited.

Glomus jugulare tumours are histologically similar to tumours of the carotid body but have their origin in the small or microscopic glomus bodies in or beneath the petrous bone. These bodies occur most frequently in the adventitia of the jugular bulb on the cochlear promontory closely related to the tympanic branch of glossopharyngeus as it passes through the canal between the jugular fossa and the middle ear, and along the auricular branch of the vagus nerve (GUILD 1953). Tumours may arise in any of these bodies their clinical manifestation differing with their site of origin, direction of growth and stage at which they are first detected. The emphasis in this paper is on

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REFERENCES

- ANDERSEN P T The lenticulo striate arteries and their diagnostic value. A preliminary report. *Acta radiol* 50 (1958) 84
- BEVOR C T The cerebral arterial supply. *Brain* 30 (1907) 403
- FEAR A The normal cerebral angiogram. Charles C Thomas Springfield Ill 1951
- GALLOWAY J R and GREIFF J The medial and lateral choroid arteries. An anatomic and roentgenographic study. *Acta radiol* 53 (1960) 353
- HAYMAKER W Handbuch der speziellen pathologischen Anatomie und Histologie (Henke-Lubarsch) Springer Verlag, Berlin Göttingen Heidelberg 1957
- HUBNER A Zur Topographie der Ernährungsgebiete der einzelnen Hirnarterien. *Zbl med Wiss* 52 (1872), 817
- JOHANSON C The central veins and deep dural sinuses of the brain. An anatomical and angiographic study. *Acta radiol Suppl* (1954) 107
- KODAMA Y Atlas of human anatomy. Vol V. Kanehara-shuppan Tokyo Japan 1956
- KRAUSHAUSEN H und RICHTER H R Die zerebrale Angiographie Georg Thieme Verlag, Stuttgart 1952
- LINDBLOM K A roentgenographic study of the vascular channels of the skull. *Acta radiol Suppl* (1936) 30
- LINDENBERG R Handbuch der speziellen pathologischen Anatomie und Histologie (Henke-Lubarsch) Springer Verlag Berlin Göttingen Heidelberg 1947
- SJOGREN S I The anterior choroidal artery. *Acta radiol* 46 (1956) 143
- SUZUKI M Studies on the distribution of the basal branches of human cerebral arteries. (Japanese). The Sapporo Medical Journal 19 (1961) 307
- TESTUT I Traité d'anatomie humaine. Gaston Doin & Cie Paris 1929
- WEISBERG G The recurrent artery of Heubner and the arteries of the central ganglia. *Acta radiol Diagnosis* (1963) 949

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Fig. 1. Tumour arising in the right jugular foramen. Enlargement of the right jugular foramen and adjacent bone destruction visible in the conventional semi-lar view (top) but better shown in the tomograms.

those tumours which produce neurological symptoms and signs rather than those which present primarily as otological problems.

The commonest neurological symptoms are those due to unilateral 9th, 10th, 11th and 12th cranial nerve palsies caused by a tumour growing in the jugular foramen. The middle ear is often involved, producing deafness,



Fig 2 Normal antero-posterior tomogram (upper view) for comparison with the abnormal tomogram below. Right side: tumour causing bone destruction at the jugular foramen and at the jugular tubercle anterior condylar canal and inferior medial part of the petrous bone (below).

tinnitus and facial palsy, and a sanguineous discharge will occur if the tumour protrudes through the tympanic membrane or invades the external auditory canal. Occasionally the presenting features are those of a posterior fossa tumour but careful clinical assessment will usually reveal an associated jugular foramen syndrome. Two patients in the author's experience suffered a subarachnoid haemorrhage, in one it was the presenting symptom. It is those patients in whom neurological symptoms predominate or precede the otological which are a diagnostic challenge to the radiologist.

The diagnostic radiologist's responsibility in the investigation and treatment of glomus jugulare tumours is fourfold:



Fig. 3 a) Normal oblique tomogram with the head turned 15° to the opposite side b) Oblique tomogram of patient shown in fig. 2 demonstrating more clearly the destructive bone changes in the jugular tubercle and anterior condylar canal

1 To establish or confirm the diagnosis, especially in those cases which present primarily as neurological problems. This should prevent precipitate neurosurgical exploration of an essentially non-operable tumour and thus avoid the occasionally fatal haemorrhagic sequelae.

2 To determine the site and size of the tumour. This is not only to assess the field size for radiotherapy but to help decide on the need for prophylactic ventriculo-cisternostomy before commencing treatment in cases with raised intracranial pressure.

3 To exclude other neoplasms involving the petrous bone and the presence of primary or secondary neoplasms elsewhere.

4 To assess the results of treatment.

These objectives can be achieved only by utilising all the available neuro-radiological techniques.

Conventional radiography. The bone changes are frequently diagnostic in themselves but do not show the size of the tumour. Standard views of the skull and special views of the petro-mastoids should be taken, including tomograms. Emphasis should be placed on the semi-axial view and on the modified submento-vertical view advocated by ERASO (1961) to show bone changes due to a tumour growing in the jugular foramen. The foramina are



Fig 4 Glomus jugulare tumour. A subtraction print of one of a lateral series of a common carotid angiogram. The tumour is calculated with and below the petrous bone is more clearly seen than on the original film.

frequently unequal in size and there must be additional evidence of bone destruction for unilateral enlargement to be of significance. Antero-posterior tomograms of the petrous bone are usually informative (Fig 1) and oblique tomograms may also be instructive. With the head turned 45° towards the unaffected side the jugular foramen and anterior part of the condylar canal are well seen (Fig 3a). If the head is turned to the affected side a good view is obtained of the whole of the anterior condylar canal and of the jugular fossa and foramen.

As the tumour enlarges bone destruction may be seen in the jugular tubercle, occipital condyle and condylar fossa (Fig 2 lower view and Fig 3b).

Anterior extension of the tumour may cause erosion in the basi-occiput, temporo-mandibular joint or floor of the middle fossa. Extension upwards will produce diffuse destructive changes in the petrous bone and elevation of the petrous ridge and tegmen tympani. These changes have been described and illustrated in detail elsewhere (COVA *et coll.* 1952; HOLESH 1955; KEMP HARPER 1957; HAWKINS 1961).



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Fig 3 a) Normal oblique tomogram with the head turned 15° to the opposite side b) tomogram of patient shown in fig 2 demonstrating more clearly the destructive bone changes at the jugular tubercle and anterior condylar process

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Fig. 5. Vertebral angiography (same patient as in fig. 4. a) Arterial phase b) Venous phase. The intra cranial part of the tumour is demonstrated but not the intra osseous part. (The common carotid angiography failed to demonstrate the intra cranial extension of the tumour.)

Angiography. It is important to perform angiography to try to demonstrate the position and size of these tumours which are normally highly vascular. The glomerula from which they arise have a blood supply mainly from the tympanic branch of the ascending pharyngeal artery. The structures commonly invaded by the tumour are supplied by small tympanic and meningeal arteries which arise from branches of the external carotid artery, and similar small vessels arising from the internal carotid and vertebral arteries anastomose with them.

Hence common or external carotid angiography should be first performed in an attempt to demonstrate a pathologic tumour circulation. This will show either intense contrast filling of fine tumour vessels in and around the area of bone destruction or pooling of the contrast medium in vascular lakes within the tumour. An arterio-venous shunt may be seen communicating with the jugular vein or with the transverse sinus if the vein is obstructed by tumour (HOOPER 1955). The pathological tumour circulation is better seen if the subtraction method is used (Fig. 4).

Carotid angiography may however fail to demonstrate intra cranial extension of the tumour. If this is suspected clinically, vertebral angiography should be performed since this part of the tumour may be fed by the vertebral artery or acquire a blood supply from the basilar artery (RUMENSCHEIDER et coll 1953).

Fig. 5 illustrates the findings at vertebral angiography in the patient whose carotid angiogram is shown in Fig. 4. The intra cranial part of the tumour



Fig 6 Myodil ventriculogram. Right-sided glomus jugulare tumour. The intracranial extension of the tumour is not only distorting the brain stem but is invading the fourth ventricle. The Myodil escaped from the ventricle through a false passage around the margin of the tumour.



Fig 7 Right jugular phlebogram, oblique projection, demonstrating a large part of the tumour which otherwise would have been missed growing within and obstructing the upper part of the internal jugular vein.

in the posterior fossa is visible but not the intrapetrous element. The carotid angiogram showed the intra- and intrapetrous tumour but failed to demonstrate this intracranial extension.

Ventriculography. Air or Myodil ventriculography are alternative methods of showing posterior fossa extension and although perhaps a less sensitive index of the size and position of the tumour may demonstrate brain stem distortion and the presence of an obstructive hydrocephalus. Fig 6 shows one stage of a ventriculography with Myodil. The intracranial extension of a right-sided tumour is not only producing brain stem distortion but is invading the fourth ventricle. The Myodil escaped from the ventricle through a false passage around the margin of the tumour.

Jugular phlebography. Jugular phlebography has been little used in the investigation of these tumours. There are at least four recorded cases where intraluminal extension into the internal jugular vein has been demonstrated at autopsy (TERRACOL & GUERRIER 1951, BICKERSTAFF & HOWELL 1953, HENSON

et coll 1953) Theoretically it would seem reasonable to suppose that tumours arising in the jugular fossa may produce extrinsic or intrinsic filling defects in the jugular vein by virtue of their close apposition to it. In the one case where this radiological technique was used, a large intraluminal extension was demonstrated growing down from the jugular fossa and occluding this vessel (Fig. 7). This part of the tumour was not shown by angiography and would have been missed had phlebography not been carried out.

This investigation could be used in early cases to establish or confirm the diagnosis and in more advanced cases to demonstrate fully the extent of the tumour. The relationship, if any, between intraluminal extension of a tumour and the occurrence of distant metastases might be clarified if this technique were more widely practised.

Discussion

There should be little difficulty in correctly diagnosing the majority of cases of glomus jugulare tumour. A polyp accessible to biopsy will be present in the external or middle ear in most patients. This diagnostic method carries some risk from haemorrhage if the operator has not been forewarned of the probable nature of the polyp. A radiological diagnosis can be arrived at in most cases, both otological and neurological in presentation, if a full examination of the skull is first carried out. This is only part of the radiological responsibility, and angiography, ventriculography and phlebography may all be necessary to confirm the diagnosis and to obtain the information which should be available before commencing treatment. The two conditions that are most likely to give rise to difficulty are meningiomas arising in the region of the sigmoid sinus, and tumours of the glomus intracraniale (BERK 1961). If however, all the techniques available to the radiologist are made use of even these should not cause insuperable difficulties in differentiation from tumours of the glomus jugulare.

Addendum in proofs

Since submission of this paper two publications (GEJROT 1964; GEJROT & LAURÉN 1964) have established the value of jugular phlebography in the diagnosis of glomus tumours in the jugular region. Of 12 patients with glomus jugulare tumours examined by retrograde phlebography 9 had filling defects within or obstruction of the internal jugular vein at the base of the skull. The three cases with negative findings were neurologically normal and it seems probable that in these instances the tumours had their origin in the tympanic bodies in the middle ear rather than in one of the glomera of the jugular bulb. There was no reported evidence of distant metastases in those patients with intraluminal extension of the tumour in the internal jugular vein.

SUMMARY

The part that radiology can play in the investigation of glomus jugulare tumours has not always been fully exploited. The importance of radiological investigation of patients presenting primarily as neurological problems is emphasised. Some of the results of investigation of these tumours are described and illustrated.

ZUSAMMENFASSUNG

Die Möglichkeiten die die Radiologie bei der Untersuchung von Tumoren des Glomus jugulare bietet sind nicht immer voll ausgenutzt worden. Die Wichtigkeit der Röntgenuntersuchung von Patienten mit primär neurologischen Problemen wird hervorgehoben. Einige Untersuchungsergebnisse dieser Tumoren werden beschrieben und illustriert.

RÉSUMÉ

On n'a pas toujours exploité à fond les possibilités de la radiologie pour le diagnostic des tumeurs du glomus jugulaire. L'auteur insiste sur l'importance de l'examen radiologique pour des malades qui se présentent au premier abord comme des cas neurologiques. Il décrit et donne en exemple les résultats d'examens de ces tumeurs.

REFERENCES

- ALFORD B. H. and GUILFORD F. R. A comprehensive study of tumours of the glomus jugulare. *Laryngoscope* 72 (1962) 765.
- BERA M. E. Chemodectoma of the glomus intravagale. A case report and review. *Clin. Radiol.* 12 (1961) 219.
- BICKERSTAFF E. R. and HOWELL J. S. The neurological importance of tumours of the glomus jugulare. *Brain* 76 (1953) 576.
- COVA L. L., MEDA P. and VICELLI A. F. I tumori glomici timpano-giugulari. *Arch. ital. Otol.* 63 (1959) 568.
- FRASER J. I. Roentgen and clinical diagnosis of glomus jugulare tumours. *Radiology* 77 (1961) 75.
- LEJROY T. Jugular syndrome. *Acta Otolaryng.* 57 (1964) 450.
- and LACRÉN T. Retrograde jugularography in diagnosis of glomus tumours in the jugular region. *Ibid.* 57 (1964) 131.
- CHILD S. R. The glomus jugulare: a non-chromaffin paraganglion in man. *Ann. Otol.* St. Louis 62 (1953) 1045.
- HAYKIN T. D. Glomus jugulare and carotid body tumours. *Clin. Radiol.* 12 (1961) 199.
- HENSON R. A., CRAWFORD J. V. and CAVANAGH J. B. Tumours of the glomus jugulare. *J. Neurol. Neurosurg. Psychiat.* 16 (1953) 127.
- HELESKI S. Diagnosis of tumours of the glomus jugulare. *Lancet* i (1955) 169.
- HOOPER R. S. The glomus jugulare tumour. Clinical and radiological features. *J. Fac. Radiol.* Lond. 2 (1955) 77.

- KEMP HARPER R. A. Glomus jugulare tumours of the temporal bone. *J. Intern. Radiol.* (Lond.) 8 (1957) 325.
- RIFMENSCHNEIDER P. A., HOOPER G. D., BREWER D., JONES D. and FICKER A. Roentgenographic diagnosis of tumours of the glomus jugularis. *Amer. J. Roentgenol.* 69 (1953) 59.
- ROSENWASSER H. Cytoid body tumour of the middle ear and mastoid. *Arch. Otolaryng.* (Chicago) 41 (1945) 64.
- TERRACOL J. and GUERRIER Y. Les tumeurs du glomus jugulaire. *Montpellier med.* 41—2 (1951), 289.

HEMODYNAMIC RESPONSES IN THE CEREBRAL VESSELS TO ANGIOGRAPHIC CONTRAST MEDIA

by

SADEK K. HILAL

Extensive experimental investigations have been carried out in dogs to study the hemodynamic changes associated with the intra arterial injection of contrast media

Material and methods

The material consisted of 66 mongrel dogs weighing between 21 and 28 kg. Eleven of these dogs represent technical failures and are not included in the reported results. Twenty seven dogs were studied for the reactions due to the injection of contrast material in the femoral artery. The changes associated with carotid injection were observed on the remaining 28 dogs. Four of these 28 had craniotomy and middle cerebral artery catheterization. Eight dogs provided the material for the determination of the least convulsive dose and finally, the last 16 dogs of this group had exposure of the carotid artery and its branches in the neck for various experiments. Intravenous nembutal was used for the induction and maintenance of anesthesia.

The blood pressure was measured with the Statham strain gauge model P 23 DE. These strain gauges have a small volume displacement and a high frequency response. The flow was measured with the square wave electromagnetic flow meter manufactured by Avionics. This unit has a frequency response of 150 cps and proved to have satisfactory long term stability.

- KEMP HARPER R. A. Glomus jugulare tumours of the temporal bone *J Fac Radiol (Lond)* 8 (1957) 325
- RIEMENSCHNEIDER P. A. HOOFLE G. D. BREWER D. JONES D. and FCKER A. Roentgenographic diagnosis of tumours of the glomus jugularis *Amer J Roentgenol* 69 (1953) 59
- ROSENWASSER H. Carotid body tumour of the middle ear and mastoid *Arch Otolaryng (Chicago)* 41 (1945) 64
- TERRACOT J. and GUIRRIER Y. *Les tumeurs du glomus jugulaire* *Montpellier med* 41—2 (1951), 289

to prevent reflux of contrast medium in the common carotid. Repeated injections were necessary to produce this effect. Five ml of material were injected at a constant rate in a three second period. After an interval of five seconds another 5 ml were injected. These 5 ml injections were repeated until generalized seizures occurred. Twenty minutes of recovery time were allowed and the same contrast was re-injected using again multiple 5 ml injections. The last injection however was reduced. This scheme allowed the determination of the least amount producing convulsions. The least convulsive dose of the other compounds was determined in the same way. Then the test was repeated on all these materials in the same order as on the first run. Two values were thus obtained for each contrast medium. The basic order of injection of these contrast media was changed from dog to dog so that every compound had an equal chance to be the first injected. Often the first seizure was difficult to initiate and a slightly larger dose than usual was required to produce the convulsion. After this first seizure the animal responded consistently.

Results

The hemodynamic changes resulting from the injection of contrast media in the carotid artery can be subdivided into two groups

I Changes resulting from the mechanical interference with the blood flow

This group of circulatory modifications occurs as a result of the injection of any liquid material in an artery and is not specific to contrast media. Most of these changes are discussed in great detail in standard texts on the physiology of the circulation and the effect of vascular narrowing. Only two aspects of these mechanical hemodynamic changes will be described here because of the general interest they stimulate and the relative lack of information available.

Changes in the arterial pressure distal to the site of injection. Outside of the jet of injection there is no increase in the peripheral blood pressure unless the needle or the catheter used has caused a significant stenosis across which a pressure gradient can be produced and maintained. This fact has been tested experimentally by monitoring the pressure in the middle cerebral artery during a carotid injection. No rise was observed when the injection catheter passed through the thyroid artery was clear from the lumen of the common carotid artery. On the other hand when the catheter was threaded in the main artery for a distance of 1 to 2 cm producing a 60% narrowing a few mm Hg rise in the peripheral pressure was observed.

Changes in the blood flow in the artery injected. The blood flow rate immediately proximal to the site of injection diminishes during the injection. When contrast media are injected this decrease is roughly equal to the injection rate. When this injection rate is faster than the local blood flow the contrast medium will run

to. Certain precautions were taken to assure accurate flow measurement. These precautions include the choice of a properly fitting probe for each studied artery, the repeated zero calibration by arterial occlusion, the complete insulation of the animal and all attached instruments with rubber sheets and rubber binders and using only one grounding point. This point was chosen to be the ground electrode of the flow probe. For the measurement of the internal carotid flow, two special probes were available having 1.0 mm and 0.75 mm diameter lumen respectively.

Exposure and catheterization of various arteries in the head and neck. The common carotid was exposed in the neck through a ventral incision. A No. 19 teflon catheter was threaded in a retrograde direction through the proximal segment of the cut thyroid artery until its tip was flush with the origin of this artery from the common carotid without projecting into the lumen of the main vessel to avoid impeding the flow through the common carotid. The electromagnetic flow meter probe was positioned on the common carotid proximal to the origin of the thyroid artery so that the injected material did not pass within the field of the probe. In certain cases the internal carotid was exposed for a distance of 2 to 3 cm beyond the bifurcation carefully avoiding excessive dissection of the carotid sinus area. In these large dogs there was enough room to position the flow meter probe on the internal carotid.

The lingual artery was exposed as distally as possible in the submandibular triangle. The artery was cut and a catheter was introduced into the proximal segment for pressure recording and another catheter passed into the distal segment for contrast medium injection.

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Results

The hemodynamic changes resulting from the injection of contrast media in the carotid artery can be subdivided into two groups

1 Changes resulting from the mechanical interference with the blood flow

This group of circulatory modifications occurs as a result of the injection of any liquid material in an artery and is not specific to contrast media. Most of these changes are discussed in great detail in standard texts on the physiology of the circulation and the effect of vascular narrowing. Only two aspects of these mechanical hemodynamic changes will be described here because of the general interest they stimulate and the relative lack of information available.

Changes in the arterial pressure distal to the site of injection. Outside of the jet of injection there is no increase in the peripheral blood pressure unless the needle or the catheter used has caused a significant stenosis across which a pressure gradient can be produced and maintained. This fact has been tested experimentally by monitoring the pressure in the middle cerebral artery during a carotid injection. No rise was observed when the injection catheter passed through the thyroid artery was clear from the lumen of the common carotid artery. On the other hand when the catheter was threaded in the main artery for a distance of 1 to 2 cm producing a 60% narrowing a few mm Hg rise in the peripheral pressure was observed.

Changes in the blood flow in the artery injected. The blood flow rate immediately proximal to the site of injection diminishes during the injection. When contrast media are injected this decrease is roughly equal to the injection rate. When this injection rate is faster than the local blood flow the contrast medium will run

15. Certain precautions were taken to assure accurate flow measurement. These precautions include the choice of a properly fitting probe for each studied artery, the repeated zero calibration by arterial occlusion, the complete insulation of the animal and all attached instruments with rubber sheets and rubber binders and using only one grounding point. This point was chosen to be the ground electrode of the flow probe. For the measurement of the internal carotid flow, two special probes were available having 1.0 mm and 0.75 mm diameter lumen respectively.

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where the electromagnetic flow meter probe was placed proximal to the injection site (Figs 2 and 3)

II *Changes resulting from the pharmacological activity of the contrast media*

The nature of the reactions to the contrast medium depends on the site of injection. Although this presentation is concerned mostly with carotid angiography, a brief description of the changes associated with femoral angiography will be given as a frame of reference.

Femoral angiography The injection of a contrast agent in the femoral artery results in a marked increase of the blood flow rate to about 2 to 5 times the original rate (Fig. 1). This local increase in flow is associated with a drop in both the diastolic arterial pressure and the pulse pressure distal to the site of injection. These changes are due to severe vasodilation. No variation is observed in the central systemic pressure. This reaction can be produced by the injection of hypertonic solutions of sodium chloride, glucose or urea and is not specific to contrast agents. On the basis of this observation, the author has developed a quantitative test for the measurement of the irritating effect of various contrast media compared with standard solutions of sodium chloride. The area under the peak of the flow curve is measured accurately and is used as the basis of this comparison. The detailed description of this test will be published separately.

Carotid angiography These results are obtained from the analysis of 282 injections of contrast media and hypertonic solutions in 20 dogs. Unlike the injections in the femoral artery, carotid angiography produces important changes in the systemic blood pressure. Another significant difference is the fact that the vasodilation seen in the femoral territory is only observed in the external carotid bed while the intracranial vessels do not show any vasodilation.

CHANGES IN THE CAROTID FLOW PATTERN **a. Common carotid flow** When a contrast medium is injected in the common carotid artery the blood flow monitored proximal to the site of injection will show first a drop in the flow rate coinciding with the injection (Fig. 2). This is followed by another drop which occurs during the first systemic hypotensive phase. If this hypotension is severe the flow can actually drop to the zero level for one or two seconds as seen in Fig. 10 with sodium diatrizoate. The grave clinical significance of this marked decrease in the blood flow becomes more obvious when it is realized that it occurs in all four main cerebral trunks because it results from a central drop in pressure. Following this drop there is a marked increase in the common carotid artery flow reaching 3 to 4 times the original rate in 15 to 25 seconds after the beginning of the injection to return to the base line in 100 to 130 seconds. This increase in the common carotid flow rate is strictly due to vasodilation of the external carotid. It is important to note

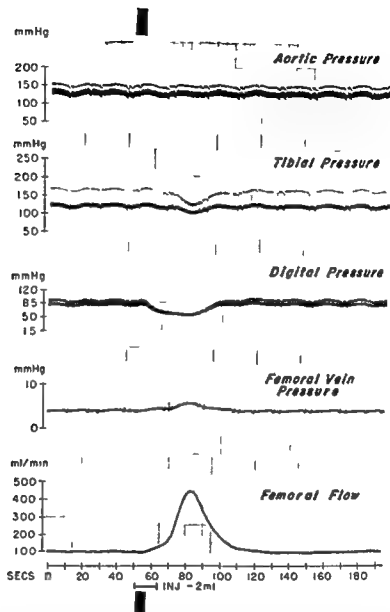


Fig 1 Injection of 2 ml of Hypaque 50% in femoral artery of canine hind limb at rate of 10 ml per minute. A 150% increase in flow rate occurs. No change seen in aortic pressure. A drop in the tibial artery and digital artery pressure associated with a decrease of the pulse pressure is observed during the peak of increased flow. Mild increase in femoral vein pressure.

in a retrograde direction. This is the usual clinical angiographic situation when peripheral vessels are studied. The contrast medium is usually administered at twice the local flow rate for one second. This decrease or reversal in the blood flow during the injection is seen on all the flow curves presented in this report.

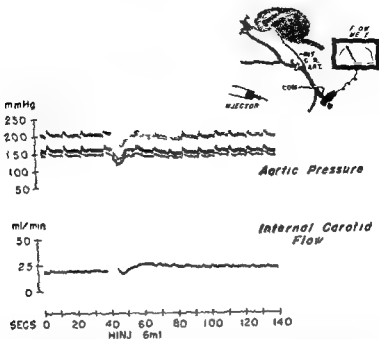


Fig 3 The external carotid is ligated and the flow probe is positioned on the common carotid. Hypaque 50 is injected in the common carotid. The aortic pressure curve shows first hypotensive phase followed by a hypertensive peak and then second hypotensive phase. The internal carotid flow curve shows reversal of the flow during the injection then a drop coinciding with the first hypotensive phase then a small rise during the hypertensive peak after which the flow curve comes down slowly to the original level. This flow curve should be compared with the common carotid flow in fig 2.

combinations are shown in Figs 2, 3, 5, 6 and 7. In the experiment shown in Fig 3 the injection was made in the common carotid artery as usual through the thyroid artery catheter and the flow probe was positioned on the common carotid proximal to the point of injection. The external carotid was ligated. A total of 58 injections in six dogs were studied. Two to six ml were injected in 1 to 2 seconds. The flow curve shows the initial drop in the flow rate due to the injection then a second drop which coincides with the fall in the systemic blood pressure and the bradycardia. Then as the blood pressure rises slightly the internal carotid flow increases correspondingly. The return of the flow curve to the original baseline usually follows the systemic blood pressure curve. Often however this return is slightly delayed and shows a 10% to 13% increase of internal carotid flow that is not justified by changes in the

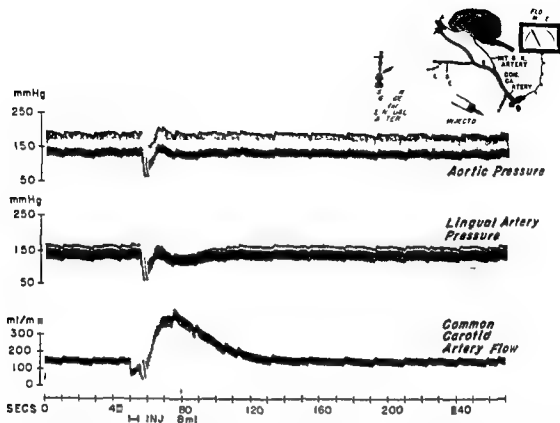


Fig. 2 Injection of 8 ml of Hypaque 30% in common carotid artery. The aortic pressure shows first hypotensive phase with bradycardia to occur about 4 seconds after beginning of injection. During this phase the diastolic pressure drops from 120 mm Hg to 60 mm Hg. This is followed by a slight hypertensive peak where the systolic pressure reaches 210 mm Hg. A mild second hypotensive phase follows where the diastolic pressure drops to 115 mm Hg from a baseline of 120 mm Hg. Lingual artery pressure reflects changes seen in systemic blood pressure and shows an additional drop during peak of increased flow resulting from vasodilation. Decrease in pulse pressure during this vasodilation. The common carotid flow curve shows first the drop occurring during the injection followed by the drop due to the bradycardia and then a sharp increase resulting from the vasodilation of the external carotid vessels.

that coinciding with the peak of the flow curve there is a drop in the lingual artery pressure associated with a decrease of the pulse pressure as seen in the vasodilation due to femoral angiography.

b Internal carotid flow. The study of the effect of the contrast media on the cerebral circulation is faced with two problems. The first is the measurement of the cerebral flow, and the second is the delivery of the contrast agent specifically to the cerebral vessels without contaminating the external carotid circulation. In the following set of experiments a variety of injection sites have been used and the blood flow has been monitored at various points. The various

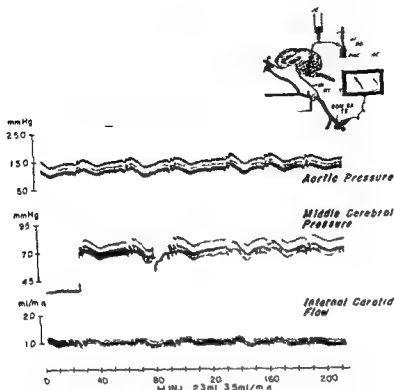


Fig 5 Selective injection of Hypaque 50 in middle cerebral artery through the catheter used for pressure recording. The middle cerebral pressure curve is interrupted during the injection. No change seen in the flow curve that would indicate cerebral vasodilation. No change in the systemic pressure.

In order to simulate the clinical situation the experiment shown in Fig 7 was performed. No artery was ligated and no craniotomy was performed. The internal carotid flow was measured with a probe positioned on the internal carotid artery. The curve shows a sharp peak occurring during the injection due to the flow of the material through the lumen of the probe. This is followed by a marked decrease corresponding to the drop in the lingual artery pressure resulting from the vasodilation in the external carotid field. This drop in the flow is due to redistribution of the blood from the unchanged intracranial vessels to the extremely dilated extracerebral vascular bed.

Only during convulsions has the internal carotid flow been observed to increase to about twice the original level. This is also associated with a drop in the middle cerebral artery pressure and narrowing of the middle cerebral



Fig. 4 Roentgenogram of head of dog showing catheterization of a branch of the middle cerebral through a craniotomy with a retrograde injection of contrast material

systemic blood pressure. This is thought to represent spillage of the contrast medium in the external carotid circulation. Since the external carotid is ligated a portion of the internal carotid flow supplies the external carotid bed and this portion of the flow will increase in case of external carotid vasodilation. If the vertebral artery on the same side of the injection is tied the internal carotid flow curve is seen to follow the systemic blood pressure more faithfully. This is due to the fact that the internal carotid blood is then diverted mostly to the brain to compensate for the vertebral occlusion.

To overcome the problem of external carotid contamination the contrast medium has been injected in the middle cerebral artery through a catheter threaded in a retrograde direction as shown in Figs 4, 5 and 6. The internal carotid flow does not show any change. Also, if cerebral vasodilation had occurred a drop in the middle cerebral artery pressure would have been registered together with a narrowing of the pulse pressure. The curves shown in Fig. 6 demonstrate the result of middle cerebral artery injection given at a fast rate. The drop in the middle cerebral artery pressure registered is not the result of cerebral vasodilation because it coincides in time and is equivalent in magnitude to the drop in the aortic pressure, and most important, there is no decrease in the pulse pressure. Twenty middle cerebral injections have been performed in four dogs all of them showing similar results.

larger doses of the contrast medium than those necessary to produce the vasodilation. The injection of 2 to 3 ml in one to two seconds is required to produce the reactions. This represents an injection rate similar to the flow rate in the carotid artery resulting in exposing the receptors of these reflexes to the full concentration of the material injected. The author has divided the disturbance in the systemic pressure into two phases.

a. The first hypotensive phase. This phase (Figs 2, 7, 8) is characterized by a marked drop in the systemic blood pressure secondary to severe bradycardia which can even reach the point of asystole for two seconds (Fig. 10). It starts about four seconds after the beginning of the injection, reaches its maximum by the fifth or seventh second and the normal heart rate is resumed by the twelfth to the fifteenth second after the beginning of the injection. With the increase of the dose of contrast media the bradycardia will appear sooner last longer and will be more marked. It is believed that the hypotension is secondary to the bradycardia since the systemic pressure curve in the time corresponding to the first hypotensive phase has shown bradycardia without hypotension while no hypotension has been seen without bradycardia. Occasionally a hypertensive peak is seen at the beginning of the reaction during the first one or two cardiac beats after the onset of the bradycardia (Fig. 7, Fig. 8 on the 48 gm NaCl/L tracing). This peak seems to result from a transient increase in the cardiac stroke volume associated with the bradycardia. A similar peak can be seen at the end of this first hypotensive phase before the heart resumes its normal rate (Figs 2, 7 and 9).

Bilateral vagotomy or atropine (0.5 mg/kg) will completely block this bradycardia indicating that the motor limb of this reflex is through the parasympathetic vagal supply to the heart. The receptor limb of this reflex is more difficult to track. Two types of experimental procedures have been conducted in order to determine the site of the receptors. In the first procedure repeated injections are made in the internal carotid artery through a catheter threaded up the common carotid artery. Only one minute is allowed to lapse between injections. The bradycardia occurring with each injection diminishes in severity indicating that the sensitivity of the receptors involved is decreasing. After three or four injections as the reflex becomes markedly attenuated, an additional injection is made in the common carotid artery. A severe bradycardia is obtained indicating that a new set of receptors are being exposed. This finding rules out the idea of one receptor centrally placed in the brain and points to a more diffuse distribution of these receptors in the carotid bed. This experiment has been conducted on five dogs with identical results. In the second experimental procedure the contrast agent is injected in a retrograde direction in the middle cerebral artery to give maximum exposure of the brain

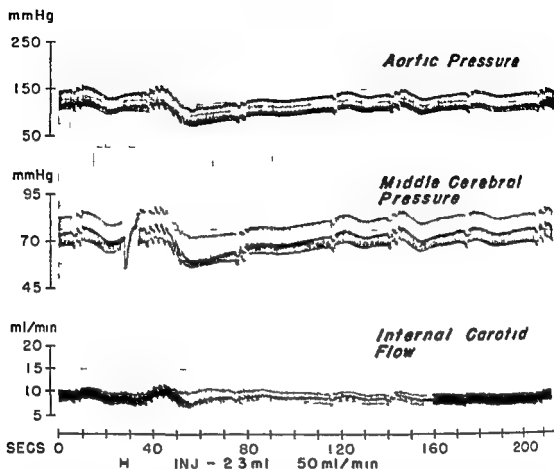


Fig 6 Same amount of material is injected in middle cerebral artery as in fig 5. A faster rate of injection is used however resulting in a better cerebral perfusion with the injected agent. Only a pronounced second hypotensive phase is produced. No bradycardia observed. If a cerebral vasodilation had taken place a narrowing of the pulse pressure would have occurred. No such narrowing is seen in the middle cerebral artery pressure tracing.

pulse pressure. This kind of change represents a true cerebral vasodilation as usually seen in other types of convulsions (12).

The vasodilation in the external carotid system, as in the femoral artery, can be produced by the injection of hypertonic solutions and does not represent a specific reaction to contrast media. The doses required to produce the vasodilation in the external carotid circulation are small, 0.5 ml of contrast medium injected slowly in a five second period is sufficient to produce the reaction. Since the flow in the canine common carotid is usually 80 ml/min the receptors of this reflex seem to respond to a 13 fold dilution of the contrast medium.

CHANGES IN THE SYSTEMIC PRESSURE These changes are produced by

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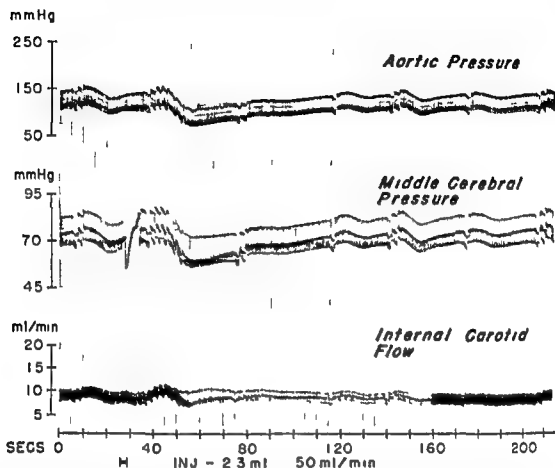


Fig. 6 Same amount of material is injected in middle cerebral artery as in fig. 5. A faster rate of injection is used however resulting in a better cerebral perfusion with the injected agent. Only a pronounced second hypotensive phase is produced. No bradycardia observed. If a cerebral vasodilation had taken place a narrowing of the pulse pressure would have occurred. No such narrowing is seen in the middle cerebral artery pressure tracing.

pulse pressure. This kind of change represents a true cerebral vasodilation as usually seen in other types of convulsions (12).

The vasodilation in the external carotid system, as in the femoral artery, can be produced by the injection of hypertonic solutions and does not represent a specific reaction to contrast media. The doses required to produce the vasodilation in the external carotid circulation are small, 0.5 ml of contrast medium injected slowly in a five second period is sufficient to produce the reaction. Since the flow in the canine common carotid is usually 80 ml/min the receptors of this reflex seem to respond to a 13 fold dilution of the contrast medium.

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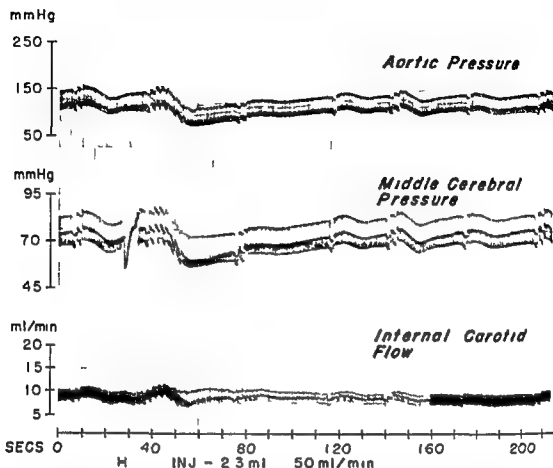


Fig. 6. Same amount of material is injected in middle cerebral artery as in fig. 5. A faster rate of injection is used however, resulting in a better cerebral perfusion with the injected agent. Only a pronounced second hypotensive phase is produced. No bradycardia observed. If a cerebral vasodilation had taken place a narrowing of the pulse pressure would have occurred. No such narrowing is seen in the middle cerebral artery pressure tracing.

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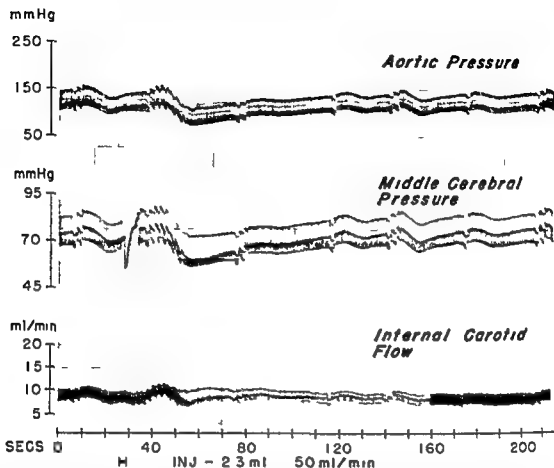


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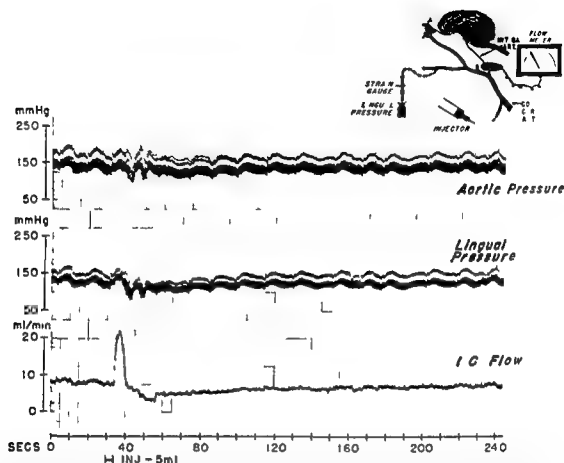


Fig 7 Changes in internal carotid flow due to common carotid injection of Hypaque 50. The two hypotensive phases are evident on the aortic pressure tracing separated by a hypertensive peak. The drop in the lingual artery pressure as evident in fig 2 coincides with the increase in the common carotid flow. On this tracing however the internal carotid flow shows a reduction during this same time which may be due to re distribution of the blood from the intra to the extracranial vessels.

Injections are also made in the distal segment of the lingual artery where the contrast medium has no way to reach the brain except in extremely dilute form. Three out of four dogs used showed no bradycardia with the middle cerebral artery injections while the fourth one showed a mild effect (Figs 5 and 6). The lingual artery injections on the other hand produced severe bradycardia in all of them (Fig 9). When a faster injection is made in the middle cerebral artery producing a more extensive exposure of the brain centers (Fig 6) hypotension without bradycardia results. This represents the second hypotensive phase described below. In the one dog where injection in the middle cerebral artery produced a mild bradycardia it is probable

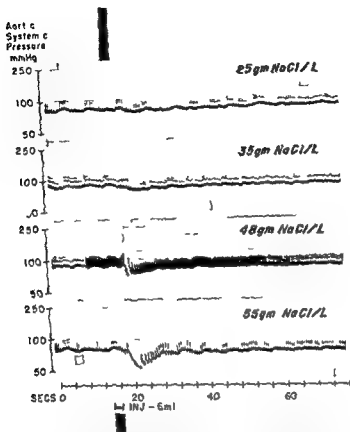


Fig 8 Disturbance of systemic pressure resulting from injection of hypertonic sodium chloride solutions in common carotid artery. The higher concentrations produce more marked hypotension.

that some of the contrast material reached the ophthalmic artery and stimulated receptors in the orbit. These findings support the view that the receptors for this reflex are rather widely distributed in the extracerebral vessels.

The first hypotensive phase can be produced by the injection of hypertonic NaCl solution and hypertonic glucose. The higher concentrations produce more marked reactions (Fig 8). Consequently, it is assumed that this reflex is not specific to contrast agents and results from a variety of irritating stimuli. No detailed quantitative study of this reaction has been made for the purpose of comparing various contrast media with standard hypertonic saline solutions. It was found, however, in our experiments as in those conducted by FISCHER &

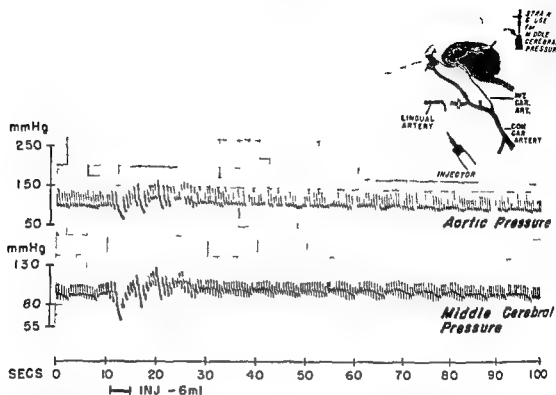


Fig 11 Tracings of the aortic and middle cerebral artery pressure indicating that the first hypotensive phase and the bradycardia can be produced by injection of 6 ml Hypaque in the distal segment of the lingual artery

ECKSTEIN (3) that the relative severity of the reactions produced by various contrast media was fairly consistent in all 16 dogs. The findings were as follows: Sodium diatrizoate (Hypaque) produced the severest reaction, sodium iothalamate (Angio Conray) a similar reaction to that of Hypaque or slightly milder, methylglucamine diatrizoate (Renografin) a reaction much milder than Angio Conray, and meglumine iothalamate (Conray) the mildest reaction of all.

b The second hypotensive phase. This phase (Figs 2, 7, 6, 10) is characterized by hypotension without bradycardia. It starts 17 seconds after the injection and reaches its lowest level at about 30 seconds. The blood pressure returns to normal at about 45–55 seconds. The drop is about 15 mm of mercury in the diastolic pressure. This reflex is less constant than the first hypotensive phase and usually requires a larger dose of contrast medium. It happened in 13 out of 16 dogs and was more obvious when the contrast medium was injected for 4 seconds at about twice the rate of flow (a total of 6–8 ml in the common carotid artery). This hypotension is produced when the injection is made into the middle cerebral artery. It seems then that it is caused by the

direct effect of the contrast medium on the brain centers. This reaction is also nonspecific in nature and can be caused by the injection of hypertonic solutions. Meglumine iothalamate (Conray) produced the mildest reaction (Fig 10).

CONVULSIVE EFFECT OF CONTRAST MEDIA Injection of contrast media in the internal carotid artery produces convulsions. This effect is discussed in this report because it is associated with cerebral hemodynamic changes as described earlier. These convulsions have been divided into two phases which seem to coincide chronologically with the two hypotensive phases described earlier.

a The phase of muscular spasm. Spasm of the neck muscles and of the body occur on the same side as the injection resulting in bending of the head and of the body to the side of the injection. This spasm occurs whether the injection is made in the common carotid or in either one of its branches. The injection of hypertonic solutions will produce it. It occurs during the first hypotensive phase and bears the same chronological relation to the injection as the bradycardia. Also as with the first hypotensive phase sodium diatrizoate produced the severest spasm while the meglumine iothalamate (Conray) produced the mildest reactions. Because of the ipsilateral nature of the spasm and the injection this reflex does not seem to be due to the effect of the injected material on the brain but rather the result of a local irritating effect.

b The phase of generalized convulsion. These convulsions start as the muscular spasm is subsiding. They are characterized by generalized bilateral seizures involving both upper and lower limbs. Their duration is variable and depends on the type of contrast medium injected. When severe they can remain for as long as two minutes. In the mild cases only one generalized myoclonic contraction may be seen.

These convulsions occur only when contrast medium is injected in the internal carotid artery in large quantities. Of all the previously described toxic effects these convulsions are the only reaction that is specific to contrast media and cannot be produced by the injection of hypertonic solutions.

Occasionally these convulsions can be caused by a single injection, however multiple injections are usually required as mentioned earlier. It appears that the seizures are the result of the damage to the blood brain barrier with the probable extravasation of minute amounts of contrast media. The first injection damages the blood brain barrier and the material injected afterwards extravasates. The least convulsive dose of the various contrast media tested in 8 dogs is as follows: Sodium diatrizoate (Hypaque) 10–12 ml; sodium iothalamate (Angio Conray) 11–13 ml; methylglucamine diatrizoate (Renografin) 16–20 ml; meglumine iothalamate (Conray) 25 ml or more.

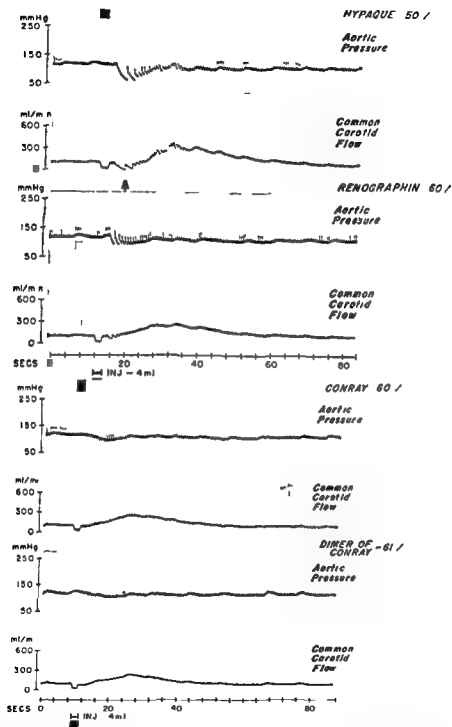


Fig 10 Comparative effect of various radiographic contrast media on systemic pressure and common carotid flow. Arrow shows drop in common carotid flow to zero level after injection of Hypaque 50. Conray dimer shows least changes in systemic pressure and least vasodilation.

Discussion

Because of the increasing frequency of the use of clinical angiography and because of the necessity to make this diagnostic procedure as safe as possible, numerous investigators have expended a great deal of effort to study the toxic effects associated with the injection of contrast media. The few references that are mentioned are only representative and by no means exhaustive. The discussion of the results presented in this work will be restricted to the changes occurring in cerebral hemodynamics.

Changes in the internal carotid flow. The results reported here correspond to the findings of SODERBERG & WECKMAN (14, 7) who postulated a redistribution of the blood flow from the intra to the extracerebral vessels with a resultant decreased pressure in the cerebral arteries.

Many investigators used a Forbes window to observe the pial vessels during carotid angiography (2, 4, 13). None of them, however, measured the systemic blood pressure with a sufficiently fast responding monometer to detect the transient hypotension and to correlate it with the variation observed in the size of the pial vessels. It is a well known fact that a sudden drop in the blood pressure causes a passive contraction of the pial vessels while a more sustained drop in the blood pressure causes dilation of these vessels (6). In carotid angiography both types of hypotensions occur and the interpretation of the results of Forbes window observation can only be accurately made in the light of a simultaneous adequate systemic blood pressure recording.

The results reported here seem to contradict the flow measurements made by LAGSTROM et al. (8, 9) who found an increase in the internal carotid flow in the dog. It is difficult to detect exactly the reason for this discrepancy. It appears, however, that the technique they used, which involves vascular surgery with multiple arterial anastomoses performed on relatively minute vessels, could affect in an unpredictable manner the normal hemodynamics. The angiographic demonstration presented in their work does not rule out the contamination of the external carotid bed with a small amount of contrast media, especially that the collateral communication between the internal and external carotid in the dog is known to be very significant. Should this contamination happen, an increase in the flow will definitely be recorded. As mentioned earlier, vasodilation can result from very small amounts of dilute contrast agent.

The two hypotensive phases. Variations in the blood pressure and heart rate resulting from carotid angiography have been reported many times (3, 5, 10, 11). The present author has shown that a marked significant decrease in the carotid flow rate occurs when the bradycardia is severe. The demonstration

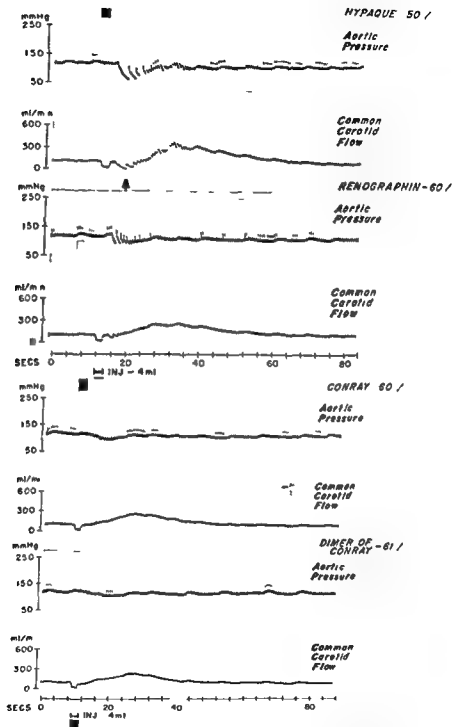


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of this transient but significant change in the flow rate was made possible because of the use of the electromagnetic flow meter

The two different components of the hypotension are described for the first time here. The identification of the different receptors involved in each case was possible because of the use of the selective injection technique where the cerebral vessels and the extracranial vessels have been injected separately.

The convulsive effect Convulsions have been observed as a result of carotid angiography (1, 7, 15). The muscular spasm seen in the laboratory experiments is also observed in a mild form in patients who very frequently bend their head slightly towards the side of the injection. The generalized seizures are seen only occasionally in the clinical situation. They seem to result from the effect of the contrast medium on the brain after the damage to the blood brain barrier. The inability of hypertonic chloride solution and hypertonic glucose to produce these convulsions can be explained in two ways: either they are unable to damage the blood brain barrier, or they are unable to produce seizures even after the vascular permeability has been damaged.

It is interesting to note that the phase of muscular spasm coincides with the bradycardia and the phase of generalized convulsions coincides with the second hypotensive phase. The first two result from the irritating effect of the contrast medium on the extracerebral structures, and the second two result from the effect of the contrast agent on the central nervous system. In addition to the chronological coincidence, the severity of the reactions in each group usually also shows a good correlation, i.e. when the bradycardia is severe the spasm is also severe. The same applies for the convulsions and the second hypotensive phase.

The determination of the least convulsive dose of the contrast media is an *in vivo* study of their effect on the blood brain barrier in a test that does not necessitate the sacrifice of the animal and thus offers the possibility of comparing various compounds on the same dog. The success of the author in obtaining repeatable results with this test is probably due to the fact that the internal carotid is ligated around the catheter. Injections of large volumes of contrast medium in this arrangement will cause the irrigation of the whole brain with the medium since the increased pressure in the vessel injected will cause a temporary stop of the blood flow from the other main arterial trunks supplying the circle of Willis.

Development of a new contrast agent From the results presented here it appears that the hypertonicity of the recently developed contrast media plays an important part in their toxicity. The chemical activity of these compounds has been considerably reduced in the last decade since the introduction of the

diatrizoate preparations followed by their methylglucamine salts and most recently the iothalamate group. The hypertonicity has been shown to be important in producing the bradycardia and in producing the external carotid vasodilation, both reactions act together to reduce the internal carotid flow. Pursuing this line of thinking the author tested the irritating effect of a new contrast agent that has a reduced tonicity for the same iodine content. This contrast agent has been prepared by the combination of two molecules of the iothalamate product by a four carbon chain link. This bisadditive has been called for convenience, the Conray dimer. The number of molecules for the same amount of iodine is thus diminished resulting in the reduction of the osmolar activity of the solution. As seen from the tracings of Fig. 10 this compound is less irritating than any available opaque agent according to these experiments. It produces less vasodilation, less systemic hypotension and no convulsions could be produced after the injection of quantities as large as 30 ml in the internal carotid artery. The muscular spasm can hardly be noticed. These findings indicate that this compound would be very suitable for carotid angiography. Clinical use however should await more extensive experimental testing.

Conclusions

The results can be summarized as follows:

- 1 Contrast media when injected intra arterially will cause vasodilation of the peripheral somatic vessels but not of the cerebral vessels. In the case of a common carotid injection this difference in effect causes a decrease of the internal carotid flow in favor of the dilated external carotid bed.
- 2 The injection in the carotid artery will produce two phases of hypotension. The first one is due to severe bradycardia resulting from the irritation of receptors in the extra cerebral carotid vessels and the second phase of hypotension is the result of the effect of the contrast media on the brain centers.
- 3 The convulsive effects of the contrast media have been studied and the author developed a technique to measure the least convulsive dose of the contrast agents. This test is felt by the author to represent an *in vivo* study of the damage to the blood brain barrier.
- 4 The importance of the hypertonicity of the contrast media as a significant contributing factor to their toxicity has been demonstrated. The author has developed another test for the quantitative evaluation of the irritating effect of the contrast media based on the comparison with standard hypertonic sodium chloride solutions in producing peripheral vasodilation.
- 5 A new contrast medium with a reduced toxicity for the same iodine content has been developed and tested with very encouraging results.

Acknowledgements

The author wishes to express his gratitude for the valuable advice and suggestions of Dr JUAN M. TRAVERA during the conduction of this study. The investigation was supported in part by Grant 5 RO 1 HE 08785 of the National Institute of Health and in part by Grant 5 T 1 NB 5298 05 of the National Institute of Neurological Diseases and Blindness.

SUMMARY

The hemodynamic changes in the cerebral vessels associated with the intra-arterial injection of contrast media at angiography have been studied in dogs.

ZUSAMMENFASSUNG

Die hemodynamischen Veränderungen der zerebralen Gefäße bei Angiographie mit intrarterieller Kontrastinjektionen wurden an Hunden studiert.

RÉSUMÉ

L'auteur a étudié sur des chiens les modifications hémodynamiques créées dans les vaisseaux cérébraux par l'injection intra-artérielle de moyen de contraste au cours de l'angiographie.

REFERENCES

1. ABBOTT K. H., GAY J. R. and GOODALL R. J. Clinical complications of cerebral angiography. *J. Neurosurg.* 9 (1952) 258.
2. BROWNE K. M. and STERN W. F. Experimental observations concerning cerebral angiography. *Arch. Neurol. Psychiat.* 71 (1954) 477.
3. FISCHER H. W. and FICKLIN J. W. Comparison of cerebral angiographic contrast by their circulatory effects. *Amer. J. Roentgenol.* 86 (1961) 166.
4. FOLTZ E. L., THOMAS L. B. and WARD A. A. JR. Effects of intracarotid diodrast. *J. Neurosurg.* 9 (1952) 68.
5. GREITZ T. A. Radiological study of brain circulation by rapid serial angiography of carotid artery. *Acta radiol.* (1956) Suppl. 140.
6. GURDJIAN E. S. and THOMAS L. M. Human pial circulation. *Arch. Neurol.* 5 (1961) 111.
7. INGVAR D. H. and SÖDERBERG U. Cerebral vasomotor tone and EEG during injection of umbradil. *Acta radiol.* 47 (1957) 185.
8. KÄGSTROM E., LINDQREN P. and TORNELL G. Changes in cerebral circulation during carotid angiography with sodium acetizoate (Triurol) and sodium diatrizoate (Hypaque). *Acta radiol.* 50 (1958) 151.
9. — — — Circulatory disturbances during cerebral angiography. Experimental evaluation of certain contrast media. *Acta radiol.* 54 (1960) 3.

- 10 LINDGREN P. Carotid angiography with triiodobenzoic acid derivatives: comparative experimental study of effects on systemic circulation in cats. *Acta radiol* 51 (1959) 353
- 11 — and TORNELL G. Blood pressure and heart rate responses in carotid angiography with sodium acetate (Triturol). *Acta radiol* 50 (1958) 160
- 12 MEYER J. S. and GOTOH F. Interaction of cerebral hemodynamics and metabolism. *Neurology* 4 (1961) 46
- 13 SCHMIDT H. W. The behavior of the pial vessels during and after the intracarotid injection of roentgen contrast media. 44 (1955) 100
- 14 SODERBERG U. and WECKMAN N. Blood pressure changes in cerebral arteries during carotid angiography with Umbradol. *Acta radiol* 50 (1958) 317
- 15 WHITELEATHER J. H. and DESAUSSE R. L. Experience with a new contrast medium (Hypaque) for cerebral angiography. *Radiology* 67 (1956) 537

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The author wishes to express his gratitude for the valuable advice and suggestions of Dr Juan M. Traveras during the conduction of this study. The investigation was supported in part by Grant 5 RO 1 HE 08785 of the National Institute of Health and in part by Grant 5 1 1 NB 5298 05 of the National Institute of Neurological Diseases and Blindness.

SUMMARY

The hemodynamic changes in the cerebral vessels associated with the intra-arterial injection of contrast media at angiography have been studied in dogs.

ZUSAMMENFASSUNG

Die hämodynamischen Veränderungen der zerebralen Gefäße bei Angiographie mit intraarterieller Kontrastinjektionen wurden an Hunden studiert.

RÉSUMÉ

L'auteur a étudié sur des chiens les modifications hémodynamiques créées dans les vaisseaux cérébraux par l'injection intra-artinelle de moyen de contraste au cours de l'angiographie.

REFERENCES

- 1 ABBOTT K. H., CAY J. R. and GOODALL R. J. Clinical complications of cerebral angiography. *J. Neurosurg.* 9 (1952) 258.
- 2 BROWN K. M. and STERN W. F. Experimental observations concerning cerebral angiography. *Arch. Neurol. Psychiat.* 71 (1954) 477.
- 3 FISCHER H. W. and ECASSTRIN J. W. Comparison of cerebral angiographic contrast by their circulatory effects. *Amer. J. Roentgenol.* 86 (1961) 166.
- 4 FOLTZ E. L., THOMAS L. B. and WARD A. A. Jr. Effects of intracarotid diodrast. *J. Neurosurg.* 9 (1952) 68.
- 5 GREITZ I. A. Radiological study of brain circulation by rapid serial angiography of carotid artery. *Acta radiol.* (1956) Suppl. 140.
- 6 GURDJIAN F. S. and THOMAS L. M. Human carotid circulation. *Arch. Neurol.* 5 (1961) 111.
- 7 INGVAR D. H. and SÖDERBERG U. Cerebral vasomotor tone and EEG during injection of umbridil. *Acta radiol.* 47 (1957) 185.
- 8 KÄGSTROM E., LINDQREN P. and TORNELL G. Changes in cerebral circulation during carotid angiography with sodium acetrizoate (Triurol) and sodium diatrizoate (Hypaque). *Acta radiol.* 50 (1958) 151.
- 9 — — Circulatory disturbances during cerebral angiography. Experimental evaluation of certain contrast media. *Acta radiol.* 54 (1960) 3.

- 10 LINDGREN P. Carotid angiography with tri iodobenzoic acid derivatives: comparative experimental study of effects on systemic circulation in cats. *Acta radiol* 51 (1959) 353
- 11 — and TORNELL G. Blood pressure and heart rate responses in carotid angiography with sodium acetrizoate (Triurol). *Acta radiol* 50 (1958) 160
- 12 MEYER J. S. and GOTOH F. Interaction of cerebral hemodynamics and metabolism. *Neurology* 4 (1961) 46
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REFERENCES

1. ABBOTT K. H., CAY J. R. and GOODALE R. J.: Clinical complications of cerebral angiography. *J. Neurosurg.* 9 (1952) 258.
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3. FISCHER H. W. and FICKSTIN J. W.: Comparison of cerebral angiographic contrast by their circulatory effects. *Amer. J. Roentgenol.* 86 (1961) 166.
4. FOLTZ E. L., THOMAS L. B. and WARD A. A. JR.: Effects of intracarotid diodrast. *J. Neurosurg.* 9 (1952) III.
5. GREITZ T. A.: Radiological study of brain circulation by rapid serial angiography of carotid artery. *Acta radiol.* (1956) Suppl. 140.
6. GURDJIAN F. S. and THOMAS L. M.: Human portal circulation. *Arch. Neurol.* 5 (1961) 111.
7. INCAR D. H. and SODERBERG U.: Cerebral vasomotor tone and EEG during injection of urobrasil. *Acta radiol.* 47 (1957) 185.
8. KÄGSTROM F., LINDGREN P. and TORNELL G.: Changes in cerebral circulation during carotid angiography with sodium acetrizate (Triurol) and sodium diatrizate (Hypaque). *Acta radiol.* 50 (1958) 151.
9. — — — Circulatory disturbances during cerebral angiography: Experimental evaluation of certain contrast media. *Acta radiol.* 54 (1960) 3.

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REFERENCES

1. ABBOTT K. H., CAVALLI J. R. and GOODALL R. J. Clinical complications of cerebral angiography. *J. Neurosurg.* 9 (1952) 258.
2. BROWNE K. M. and STERN W. T. Experimental observations concerning cerebral angiography. *Arch. Neurol. Psychiat.* 71 (1954) 477.
3. FISCHER H. W. and FRANKLIN J. W. Comparison of cerebral angiographic contrast by their circulatory effects. *Amer. J. Roentgenol.* 86 (1961) 166.
4. FOLTZ E. L., THOMAS L. B. and WARD A. A. JR. Effects of intracarotid diodrast. *J. Neurosurg.* 9 (1952) 68.
5. GREITZ T. A. Radiological study of brain circulation by rapid serial angiography of carotid artery. *Acta radiol.* (1956) Suppl. 140.
6. GURDJIAN E. S. and THOMAS L. M. Human pial circulation. *Arch. Neurol.* 5 (1961) 111.
7. INCVAR D. H. and SODERBERG U. Cerebral vasomotor tone and EEG during injection of umbradil. *Acta radiol.* 47 (1957) 185.
8. KÄGSTROM E., LINDQVIST P. and TORNELL G. Changes in cerebral circulation during carotid angiography with sodium acetrizoate (Triurol) and sodium diatrizoate (Hypaque). *Acta radiol.* 50 (1958) 151.
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FROM THE DEPARTMENT OF RADIOLOGY, COLUMBIA UNIVERSITY COLLEGE OF PHYSICIANS AND SURGEONS, AND THE RADIOLOGICAL SERVICE OF THE NEUROLOGICAL INSTITUTE, COLUMBIA PRESBYTERIAN MEDICAL CENTER, NEW YORK, AND FROM THE DEPARTMENTS OF NEUROLOGY AND RADIOLOGY OF THE UNIVERSITY OF MINNESOTA, MINNEAPOLIS, MINNESOTA, U S A

DETERMINATION OF THE CAROTID AND REGIONAL CEREBRAL BLOOD FLOW BY A RADIOGRAPHIC TECHNIQUE

by

SADAK K. HILAL, JOSEPH A. RESCH and KURT AMPLATZ

The radiographic method for the determination of the blood flow in individual carotid arteries presented here is an indicator dilution technique. The indicator is a contrast medium injected at a known constant rate in the artery of interest. The concentration of the contrast medium distal to the site of injection is a function of the blood flow rate in the artery. Higher flow rates will cause more dilution of the medium and consequently a roentgenogram of the vessel will show little optical contrast between the artery and the background because of the dilution of the contrast material. On the other hand, a small flow rate will cause a lesser dilution of the indicator and a larger optical contrast will be observed between the vessel and its background. In the method presented here the optical density is accurately measured with a densitometer using a 40 micra light beam. The dilution of the contrast medium is determined by comparing the measured optical contrast of the image of the artery against its background with the optical contrast of a set of standard tubes containing a known concentration of the contrast medium used. The



Figs 1 and 2 *Left* Determination of blood flow in common carotid artery. Tip of small teflon catheter is seen projecting from tip of angiographic catheter. *Right* Roentgenogram of cervical spine with a set of standard tubes superimposed in neck. Metallic disc on edge of field is used to determine the magnification.

flow is calculated from the dilution of the contrast agent and from the known rate of injection according to the Hamilton Stewart formula.

The determination of the regional flow will be briefly mentioned at the end of this report since it is still in the preliminary stages.

Methods

No detailed mathematical procedure will be described here as this is the subject of a separate publication. Only a brief description of the various technical aspects involved is presented.

The sensitometric curve of the radiographic film is first made. The relative exposure necessary to give the various shades of gray is thus determined. This first step is necessary for the calibration of the type of film used and the processing technique. Since the automatic processor is used, this sensitometric curve is determined only once every few months. For the blood flow determination



Fig 3 Narrowing of the internal carotid artery by 60°. Flow measurements indicated a 52% reduction as compared with opposite side

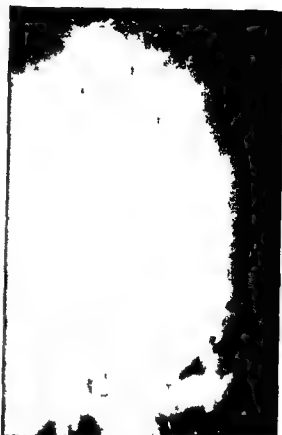


Fig 4 Carotid angiogram. Occlusion of left middle cerebral artery

the optical density of the roentgenogram taken during the constant rate injection of the contrast medium is measured in two points: one just outside the limits of the image of the artery on the film, and the second in the middle of the artery. The relative exposure corresponding to each density is determined from the sensitometric curve obtained above. Since the image of the artery on the film is lighter than its background, the relative exposure of the film corresponding to the artery is always less than the relative exposure of the background. The difference between the values of these two relative exposures represents the roentgen energy absorbed by the artery, and the ratio of these two values is the relative attenuation of the incident roentgen beam. This ratio will be called the 'attenuation factor'. Large amounts of iodinated material in the artery will cause a marked attenuation of the roentgen ray beam. The attenuation factor of the artery studied is compared with the 'attenuation factor' of various standard tubes filled with known con-

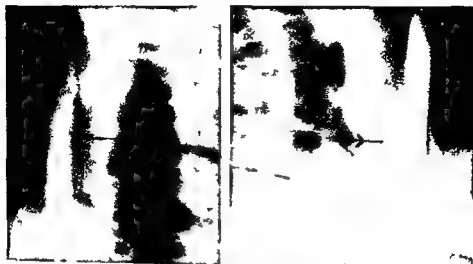


Fig 5 Relative dilution of the contrast agent by various flow rates

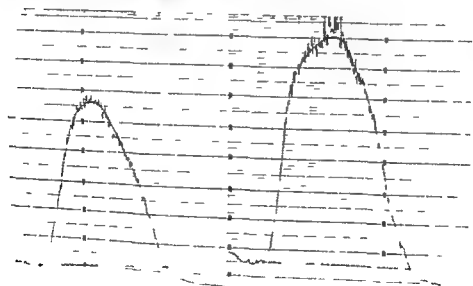


Fig 6 Densitometric tracing of the two vessels in fig 5

concentrations of contrast medium (Fig 2). From this comparison the concentration of the contrast medium in the artery is determined. Two roentgenograms are thus required: one is taken during the slow constant rate injection of the contrast medium in the artery (100 ml/min) and the other is taken with the



Fig 7 Angiogram in late arterial phase showing a tumor stain

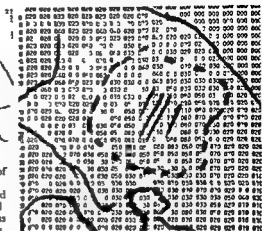
set of standard tubes superimposed on the patients without any injection (Figs 1 and 2)

Catheterization and injection technique The cases reported here were all catheterized through a subclavian puncture as reported in a previous publication (AMPLATZ 1963). After the angiogram is performed a fine teflon catheter (0.8 mm in diameter) is passed through the catheter used for angiography. The tip of this minute teflon tubing is positioned either in the common carotid artery (Fig 1) or in the internal carotid artery depending on where the flow is to be measured. No difficulty has been encountered by the author in threading this catheter up to the origin of the internal carotid artery. In addition to the fact that this small catheter allows the selective injection of the contrast medium in either the common carotid or the internal carotid, it also sprays the contrast medium into the artery since it has a closed end and a few minute side holes. This spray insures turbulent flow and adequate mixture of the blood and the indicator. In order to assure a constant rate of injection through the high resistance presented by the narrow tubing a special injector has been built. This injector has a powerful synchronous motor driving the plunger of a steel syringe.

The densitometer used for these measurements provides a projection screen on which the area of the film is viewed and in the center of this area the exact position of the scanning light beam is observed. As mentioned above, the diameter of this scanning light beam is 40 micron. The optical density is recorded on a strip chart recorder (Fig 6). From the tracing obtained the concentration of the opaque indicator in the artery is determined. (The densitometer is available from the National Spectrographic Laboratories, Cleveland, Ohio.)



Fig 8 Upper view. Digital presentation of regional flow obtained by Halal Dual Densitometer from film of fig 7. Rt. Magnified view of tumour & ea. Numbers represent volume of contrast medium expressed in fractions of ml present in a rectangular region measuring 3 mm \times 4 mm



Results

The blood flow rate has been determined on 18 patients with this method. Only three representative cases will be reported here.

The first case is a 55 year old patient with no organic neurologic disease and with normal angiography. Multiple flow measurements were made on each common carotid artery.

Rt. com. carot. flow in ml/min 547 533 533 538

Lt. com. carot. flow in ml/min 497 489 497 500 Avg 496

This case is presented to illustrate the repeatability of the results. Every figure reported here represents a separate injection and of course a separate film on which the densitometric measurements were made.

The second case is a patient with a 60% narrowing of the left internal carotid at the bifurcation (Fig 3).

Rt. int. carot. flow in ml/min 288 291 Avg 290

Lt. int. carot. flow in ml/min 139 138 Avg 139

This 60% narrowing of the internal carotid artery has caused a reduction of the flow to about 48% of the rate of flow of the opposite side.

The third case is that of a patient with an occlusion of the left middle cerebral artery (Figs 4, 5 and 6). The flow determined in each internal carotid artery is as follows:

Rt. int. carot. flow ml/min 227 301 Avg 262

Lt. int. carot. flow ml/min 179 207 147 177 Avg 176

This flow on the left side is reduced by 33% as compared with the flow on the right side.

Discussion

From the examples given it is evident that the repeated determination of the flow in the same vessel by this method provides fairly consistent results.

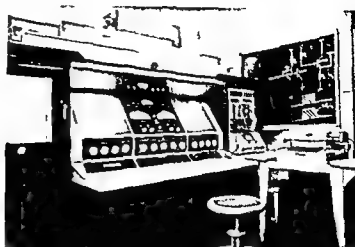


Fig. 9 General view of the Hilal Dual Densitometer

indicating that the various radiographic procedures can be adequately controlled for the purpose of this work. It is also important to note that the variation between repeated determinations is much smaller than the variation between the normal and the pathologic states.

The results obtained here for the normal carotid flow agree with the measurements made by KRISTIANSEN & KROG (1962) and TINDALL et coll (1962) using the electromagnetic flow meter. These results are also in fair agreement with those obtained by SHELLIN et coll (1948) and by NALIN (1961) using a non diffusible indicator.

Since the flow is determined in individual vessels by this method, the hemodynamic significance of a single stenosis can be assessed. This information could be important in the evaluation of a patient for vascular surgery. On the other hand, when a method that measures total cerebral flow is used the disturbance resulting from a particular localized vascular region is difficult to assess because the compensatory increase in the flow in the other functioning vessels will overshadow this disturbance.

Regional blood flow determination This procedure is still in the experimental stage and no sufficient data are available for critical appraisal. A brief description, however, is given here. As in the intracarotid injection of isotopes (FAZIO et coll 1963) the rate of renewal of the cerebral blood pool can be calculated from the measurement of the density of a particular region on all the films of an angiographic series. In the radiographic method, however, in addition to this information, we have the concentration of the indicator in the arterioles supplying the region of interest and the veins draining it. From these data the size of the regional cerebral blood pool studied can be determined and the

actual regional flow can be calculated. The apparatus necessary to make these measurements has been designed by the author and built for this purpose (Fig. 9). It consists of a dual densitometer capable of scanning two films: the first is the regional film before the injection of the contrast agent, and the second is a film from the angiographic series. Provision is made for the accurate alignment of the two films so that identical regions of the skull are scanned on the films. The signals from the photomultipliers are received by a computer which, through a process of logarithmic subtraction of the optical densities of the two films, determines the quantity of contrast medium in the area scanned. The results are presented in digital form as shown in Fig. 8. Each of these numbers represents the volume of the indicator present in a rectangular area measuring 3×4 mm. The spatial resolution obtained by this method evidently exceeds the capabilities of any isotope technique.

Acknowledgements

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ZUSAMMENFASSUNG

Eine Methode zur Bestimmung der Blutzirkulation der Carotiden wird beschrieben. Bei wiederholter Bestimmung in derselben Arterie sind die Resultate konstant und stimmen mit den Werten anderer Untersuchungen überein. Diese Methode bietet die Möglichkeit für die Bestimmung von hämodynamischen Störungen, die von einer lokalisierten Stenose her stammen. Die Technik der Messung der regionalen Blutzirkulation wird in groben Zügen beschrieben. Die Methode befindet sich noch in einem vorläufigen Stadium.

RÉSUMÉ

L'auteur présente une méthode de mesure du débit sanguin dans chaque artère carotide. Des mesures répétées dans la même artère donnent des résultats constants et en accord avec les valeurs obtenues par d'autres expérimentateurs. Cette méthode permet d'étudier les



Fig. 2. General view of the Hilal Dual Densitometer

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perturbations hémodynamiques résultant d'une sténose localisée. L'auteur décrit brièvement une technique de mesure du débit cérébral régional. Cette méthode est encore au stade préliminaire.

REFERENCES

- AMPLATZ K., RESCH J. and HILAL S.: A catheter approach for cerebral angiography. *Radiology* 81 (1963) 576.
- FAZIO C., FUSCHI C. and ACQUATI A.: Direct common carotid injection of radioisotopes for the evaluation of cerebral circulatory disturbances. A study of 116 cases by bilateral radioisotope rheoencephalography. *Neurology* 13 (1963) 561.
- HARDEN W. H., ROBERTS B., TOOLE J. I. and ROYSTER H. P.: Studies on carotid artery flow. *Surgery* 49 (1961) 251.
- KRISTIANSEN K. and KROG J.: Electromagnetic studies on the blood flow through the carotid system in man. *Neurology* 12 (1962) 20.
- NYLIN G., HEDINUND S. and RECNSTROM O.: Studies of the cerebral circulation with labeled erythrocytes in healthy man. *Circulat. Res.* 9 (1961) 664.
- SILINKIN H., HORNEL M. and KETS S.: Dynamic anatomy of cerebral circulation. *Arch. Neurol. Psychiat.* 60 (1948) 240.
- STEWART G. N.: Researches on circulation time and on influences which affect it. IV. Output of the heart. *J. Physiol.* 22 (1897) 159.
- INDALL G. T., ODOM G. L., CUPP H. B. and DILLON M. L.: Studies on carotid flow and pressure. *J. Neurosurg.* 19 (1962) 917.

EVALUATION OF CLOSED CIRCUIT TELEVISION TECHNICS IN NEUROROENTGENOLOGY

by

COLIN B HOLMAN

Closed circuit television technics are being employed in diagnostic roentgenologic practice in ever increasing numbers and in a variety of procedures. Basically these include two fundamental and different applications which at times may be combined namely as a medium of rapid, visual communication and as a method of image enhancement for improvement in diagnosis. The first category includes transmission of the fluoroscopic image to various monitoring and recording devices as well as transmission of a film image to one or more remote monitors for observation. The second category includes any applications relating to the manipulation of the roentgenographic or roentgenoscopic image in order to bring out information that might not be apparent on ordinary inspection. The most dramatic and practical application in this regard is the performing of the subtraction technic using various contrast studies. Other applications include magnification of the image and changing within limits the density and contrast of various roentgenograms. Bas relief effects can be produced for teaching purposes, and relative measuring of certain anatomic structures and locations can be done. This presentation will be limited to the second category although it is realized that the chief

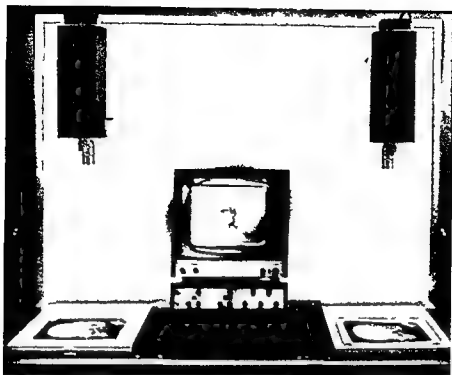


Fig 1 Closed circuit television apparatus arranged for simultaneous projection of two roentgenograms

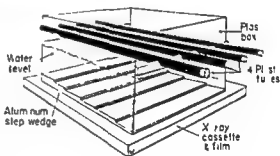


Fig 2 Phantom for studying resolution of density of various concentrations of contrast medium when projected through aluminum step wedge

applications of closed circuit television to date have been those relating to its use as a medium of communication

Image enhancement or manipulation has been a goal of photographic techniques for many years, and most if not all of these procedures have been applied at one time or another to roentgenograms. The advent of television brought yet another technique to alter various images and therefore it is reasonable to expect these methods to be adapted to radiology.

It is well known that the density and contrast of a television image may be altered within limits and this is at times a rapid, practical, and useful way of

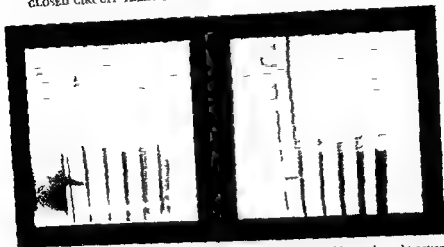


Fig. 3. Images of tubes filled with contrast material are not resolved better by subtraction as density of background increases.



Fig. 4. a) Right retrograde brachial angiography anteroposterior projection. No aneurysm visible. b) Same as subtraction. Large aneurysm arising at junction of right vertebral artery and basilar artery.

improving some roentgenograms. The success of this manipulation depends as much upon the lens system, the size of the camera diaphragm opening, and the illumination of the viewing box as it does on the television system. The television image is of a somewhat different quality from that of the film and certainly does lack some sharpness of detail. However, it is doubtful that there

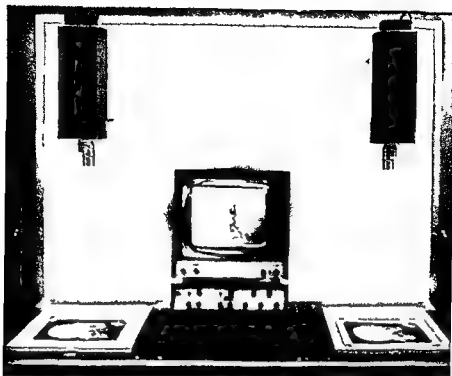


Fig 1 Closed circuit television apparatus arranged for simultaneous projection of two roentgenograms

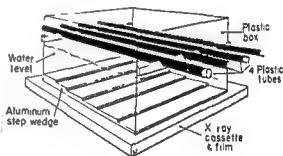


Fig 2 Phantom for studying resolution of density of various concentrations of contrast medium when projected through aluminum step wedge

applications of closed circuit television to date have been those relating to its use as a medium of communication

Image enhancement or manipulation has been a goal of photographic technics for many years, and most, if not all, of these procedures have been applied at one time or another to roentgenograms. The advent of television brought yet another technic to alter various images and therefore it is reasonable to expect these methods to be adapted to radiology.

It is well known that the density and contrast of a television image may be altered within limits and this is at times a rapid, practical, and useful way of



Fig 6 Right retrograde angiography, submentovertex projection in the same case as in figs 4 and 5. The aneurysm more evident than in the study

simultaneously (Fig 1). One camera projects the image of the prefilm. The other camera projects the disposable image of the contrast. Synchronized signals from both cameras are applied simultaneously to the same television monitor. This permits cancelling out of the elements common to each roentgenogram leaving for study an image of those elements not common to each; in this case the contrast material. The resultant subtraction image can then be changed by altering density and contrast, or it may be magnified.

It is possible to apply this method routinely in all cases of cerebral angiography in order to study more easily the structures filled with contrast, and also to identify in some cases collections of contrast material in vessels or aneurysms that are not apparent because of overlying structures on ordinary inspection of the roentgenogram. It has been successfully also to encephalography and gas myelography.

In an effort to determine experimentally whether one could expose structures that were not visible at all on a regular angiogram, the following experiment was done:

Plastic tubes of varying calibers were filled with dilute solutions of contrast material. These were placed in a water phantom on the floor of which was placed an aluminum step wedge (Fig 2). Films of the phantom were exposed with and without the opaque medium in the tubes, and the subtraction studies were made. It was evident (Fig 3) that the tubes filled



Fig 5 Same case as in fig 4 a) Right retrograde brachial angiography lateral projection Aneurysm obscured by dense bone at base of skull b) Television subtraction Aneurysm projected in anterior portion of foramen magnum

is much advantage in employing a system with more scanning lines than are used with the present commercial television systems. Optical magnification at the camera lens is a rapid, convenient and effective procedure and it is possible to achieve a high order of magnification with which to study details of structures that are difficult to observe on inspection of the original film. For example, it is possible to resolve between 200 and 300 lines per inch with a lens system and an ordinary closed circuit television system. In the case of the appreciation of tiny vessels the limiting factor does not seem to be the resolving power of the television system but the fact that the vessels, being very small, cannot contain enough contrast material to absorb photons of energy to such a degree that a visible difference from the surroundings will arise.

Reversal of the roentgen image so that a dipositive picture is obtained is also a simple maneuver with a television system. Occasionally this subjectively improves the roentgenographic image. This is the manner in which many European medical journals print their roentgenograms.

Image reversal is the key to television subtraction and it is in this field that the television system has been of most immediate help. The advantages of photographic subtraction in some examinations are well known. The principal advantage of the television method is the extreme rapidity with which high quality subtraction studies may be accomplished.

Briefly the method consists of employing two closed circuit television cameras

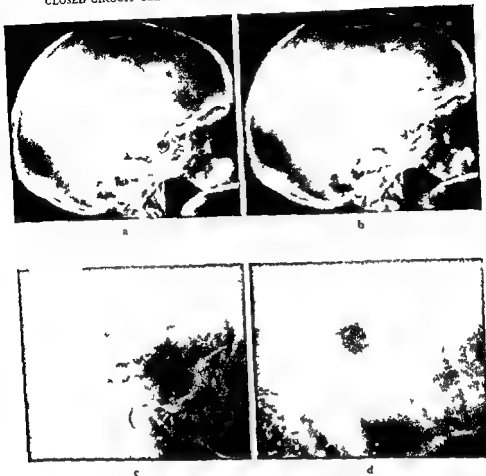


Fig 8 a) Lateral roentgenogram of head showing osteoma of tuberculum sellae b) Angiography of the common carotid artery in lateral phase lateral projection c) Television subtraction study of fig 8b showing the vascular port on of the tumor around the osteoma and extending into sella turcica d) Television subtraction study of fig 8c magnified on television monitor

Perhaps the most valuable common use of this technique is in the study of vessels that are partly hidden by structures at the base of the skull and the cervical spine such as in angiography of the vertebrobasilar system. It is possible to make the examination using any projection because the vessels will not be obscured by overlying bone. This applies to the venous sinuses as well as to the arterial supply. Similarly poorly contrast filled vascular tumors are seen with great clarity although they might be overlooked on the regular film examination.



Fig 7 a) Vertebral angiogram obtained by injection of contrast material into aortic arch. The vessels are obscured by base of skull and cervical spine. b) Television subtraction. The arteries are visible.

contrast material could not be seen any further through the step wedge with subtraction than they could without this process, thus indicating, what might be predicted, namely that the subtraction study seemed to have its merit in distinguishing contrast filled structures from other details of similar density rather than in revealing a totally new structure on the roentgenogram. This is a surprisingly valuable procedure however, because not infrequently lesions are discovered which are missed on ordinary inspection. The following examples serve to illustrate.

Fig 4a is the anteroposterior angiogram of a patient who previously had been examined twice for subarachnoid bleeding. Even a careful study of the film at the region of the foramen magnum does not reveal any particularly important abnormality. Fig 4b shows the subtraction study which demonstrated an aneurysm located at the origin of the basilar artery. The contrast medium within this aneurysm was present on the original examination but could not be seen because of overlying structures. Fig 5a shows a lateral view of the same patient. In this instance it is noted that the aneurysm again overlies the posterior fossa and is seen only on the subtraction study (Fig 5b). Fig 6 shows a submentovertical view of a third injection to prove that the aneurysm is actually present, although it obviously is not more evident than in the subtraction study.

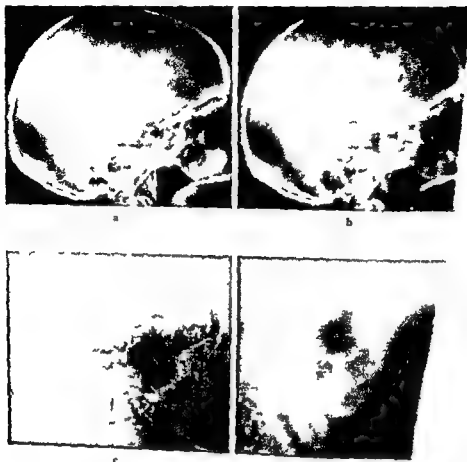


Fig 8 Lateral roentgenogram of head showing common carotid artery in different phases. (a) and (b) showing the common carotid artery in different phases. (c) and (d) showing the vascular position of the tumor. (c) and (d) are subtraction studies of (a) and (b) respectively.

Fig 7a shows the vertebral arteries obscured by the overlying cervical spine and base of the skull but in Fig 7b it is easy to see the contrast filled vessels. Fig 8 shows the possibility of enhancing a poorly contrast filled tumor. The lateral film of the head (Fig 8a) shows an osteoma arising from the tuberculum sellae. It was known that meningioma should be present in this region but the extent of the soft tissue portion could not be appreciated. Fig 8b shows the midarterial phase which does not give a clear view of the perfused tumor. The subtracted image of the same picture is shown in Fig 8c. The black, contrast filled vascular portion of the tumor extending well down into the sella turcica is apparent. Fig 8d represents a magnification also immediately done on the television monitor, giving a good view of the tumor extending into the sella turcica.

Two other uses of this apparatus warrant mention. For teaching purposes it is possible to produce high relief effects by offsetting the images of two identical roentgenograms. This is just an extension of the photographic method known for years and it normally aids in the study of roentgen anatomy.

Another use concerns the production of matching overlay studies which differ in size. Thus, for example, it is possible to match the pinet localization graph to any particular head in order to determine whether the calcification lies within the normal range. One can also superimpose the radioisotope scan film on the roentgenogram and obtain an idea of the localization of the lesion. This, of course, can be done directly, but it is sometimes of help to change the density or contrast, or both, of one or the other image to appreciate the information.

It is thought that closed circuit television systems offer great convenience as a medium of roentgenographic communication, but perhaps just as significantly, within limits, they permit certain manipulations of the roentgenographic image in order to obtain rapidly more information of diagnostic value than it is possible to obtain with direct study of the film, and it is in this field that it is suggested that more investigation should be carried out.

SUMMARY

Closed circuit television techniques enhance some radiologic examinations. Subtraction studies are easily performed with two synchronized television cameras linked to a single TV monitor. Angiographic details are more readily appreciated than when viewed on the original angiogram. Ordinary closed circuit television systems can resolve as many as 300 lines per inch so that the limiting factor is not the television system but the contrast in roentgenographic densities between tiny structures and the background.

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Geschloss ne Stromkreis Television intensifiziert einige radiologischen Untersuchungen. Subtraktionsstudien sind mit zwei synchronisierten TV Kameras die an einen einzigen TV Monitor angeschlossen sind leicht durchzuföhren. Angiographische Details treten viel deutlicher hervor als am Originalangiogramm. Das Auflösungsvermögen einer gewöhnlichen geschlossenen Stromkreis TV ist so hoch wie 300 / inch so dass die obere Begrenzung nicht durch das TV System sondern durch den röntgenographischen Kontrast von Struktur und Hintergrund gegeben ist.

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BIBLIOGRAPHY

- HANAFEE W and STOUT P. Subtraction technique. *Radiology* 79 (1962) 658.
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PRECENTRAL CEREBELLAR VEIN IN ANGIOGRAPHY

by

YUN PINC HUANG and BERNARD S. WOLF

Study of the veins of the posterior fossa is complicated by numerous variations in the course and communications of these vessels. Many of these veins are small and radiographically difficult to demonstrate. Moreover, classical anatomical descriptions of these veins are not sufficiently detailed for radiologic purposes (1, 2, 3, 6, 7, 9, 10, 11). In the radiologic literature (1, 5), only a few major veins have been noted. The purpose of this paper is to describe a specific vein rather constantly seen during vertebral angiography, which has not been clearly described in either the anatomical or the radiologic literature. Since this vein originates in the precentral cerebellar fissure, that is, the fissure between the central lobule of the cerebellum and the lingula, it may be designated as the precentral cerebellar vein or, in suitable context, simply as the precentral vein. In the injection studies performed by JOHANSSON (8), this vein is clearly demonstrated although no specific comment was made about this vessel.

On gross inspection of the intact brain only a small portion of the anterior aspect of the cerebellum behind the mid brain is visible unless the brain stem is forcibly separated from the cerebellum. When this is done a surprisingly deep cleft (Figs 1 and 2) is exposed extending downward behind the superior and middle cerebellar peduncles over the roof of the upper part of the fourth ventricle.

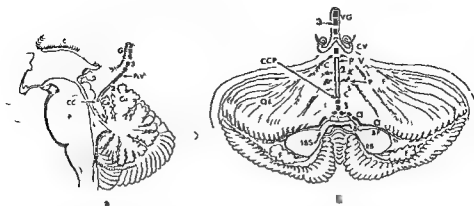


Fig. 1 a) Diagrammatic representation of posterior fossa structures in midline sagittal section. First portion (1) of precentral vein (PcV) is located in the fissure between the lingula (L) and the cerebellar lobule (Ce) in the precentral fissure. The colliculo-central point (CCP) is situated between the superior colliculus (S) in front and the central lobule (Ce) behind. Second portion of the vein (2) runs in front of the culmen. Third portion (3) runs above the apex of the cerebellum in the cisterna ambiens and enters posterior end of great vein of Galen (VG). The first portion of the vein follows a course parallel to the roof of the upper part of the 4th ventricle. Other labeled structures are: superior colliculus (S), pons (Po) and internal cerebellar vein (ICV).

b) Diagrammatic frontal representation of cerebellum with brain stem removed and margins of the anterior cerebellar notch separated. This is approximately the view achieved in a 30° oblique projection (half axial view). First portion (1) of precentral cerebellar vein (PcV) is formed by the union of symmetrical tributaries (brachial veins) from each side. The brachial veins (a and a') begin on the brachium pons (BP) and run medially over brachium conjunctivum (BC) and lingula (L) to midline. The single midline trunk runs upward behind the lingula. (The lingula has been elevated and its upper portion removed to expose this portion of the vessel.) CCP is the knee or illiculo-central point. Second portion (?) of the vein continues upward in front of central lobule (Ce) and the culmen (Cu). Third portion (3) lies above cerebellum within cisterna ambiens and behind great vein of Galen (VG). Other labeled structures: Interbranchal sulcus (IBS), inferior cerebellar peduncle or restiform body (RB), wing of central lobule (Ala), preculminate fissure (Puf), folium (F) and quadrangular lobule (QL).

tricle toward the fastigium. The central lobule of the cerebellum lies in the mid portion of the posterior wall of the cleft. The wings or alae of the central lobule lie in the posterior wall of this cleft in its lateral portion. The cleft as a whole seems to have no specific anatomical designation. Its central portion however corresponds to the precentral fissure. It would therefore seem appropriate to refer to the lateral portions of this cleft as the lateral extensions or wings of the precentral fissure. It should be noted however, that the quadrangular lobule on each side contributes to the uppermost portion of the posterior wall of this cleft as it extends forward to be applied to the posterolateral aspect of the brain stem. The superior paravermian or paramedian sulcus separates the central lobule from its wings and continues upward on each side of the culmen to the highest point or apex of the cerebellum.

The configuration of the anterior wall of the precentral fissure and its wings

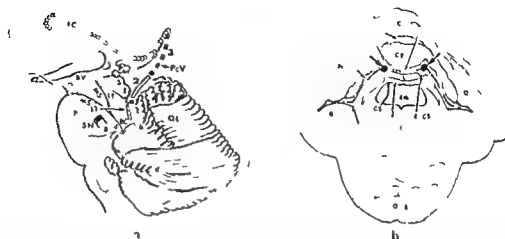


Fig. 2 a) Lateral view of posterior fossa structures with cerebellum in situ. Portions of superior vermis and adjacent hemisphere have been removed to expose brachium conjunctivum (BC) and brachium pontis (BP) as they join the cerebellum. The fine brachial vein (v) originates on brachium pontis (BP) and continues backward (v) over interbrachial sulcus (IBs) and brachium conjunctivum (BC) to lateral margin of lingula at lower end of prevermian sulcus. Small perforating veins (b) from the substance of the cerebellum join the vein in this region. The vein then runs upward and forward in precentral fissure (f) to reach the surface of cerebellum immediately behind inferior colliculi (i). A small lingular tributary (lT) from the lateral aspect of the lingula joins the vein at this level. Other labeled structures are: Basal vein (BV), lemniscal trigone (LT), fifth nerve (5N), quadrangular lobule (QL), lateral mesencephalic sulcus (LMS) and pontomesencephalic sulcus (PMS). b) Section through upper part of 4th ventricle. Plane of section is oblique to the long axis of the brain stem cutting through lower pons anteriorly and the brachia conjunctiva (BC) posteriorly. The mid portion (Pec l M) and the lateral extensions (l cel l) of the precentral fissure are shaded. The two tributaries brachial veins (unlabeled arrows) which unite to form the precentral cerebellar vein proper are located on each side of the lingula (l). The dotted lines in the lateral extensions of the precentral fissure represent the brachial veins at levels below the plane of this section. The dotted line in the central portion of the fissure represents transverse communicating channels. Other labeled structures are: Culmen (CU), central lobule (CL), preculminate fissure (Pcul), prevermian sulcus (PVS), wing of central lobule (ALA), internal velo conjunctival sulcus (IVCS), external velo conjunctival sulcus (EVCS), anterior medullary velum (AMV), interbrachial sulcus (IBs), brachium pontis (BP), quadrangular lobule (QL).

is complicated by the numerous structures in this area. In the mid portion, these include from above downward, the medial portions of the inferior colliculi, the frenulum veli, and the lingula overlying the anterior medullary velum. The lateral extensions of the precentral cerebellar fissure extend not only laterally but also anteriorly and include in the anterior wall, the lateral portions of the inferior colliculi, the superior cerebellar peduncles and more laterally the lemniscal trigones and posterior aspects of the middle cerebellar peduncles. Between the superior cerebellar peduncle (brachium conjunctivum) and the middle cerebellar peduncle (brachium pontis) on each side, there is a sulcus designated as the interbrachial or interpeduncular sulcus (not the interpeduncular fossa). The anterior part of the wing rests in this sulcus. The lateral mesencephalic sulcus leads into the interbrachial sulcus from above,

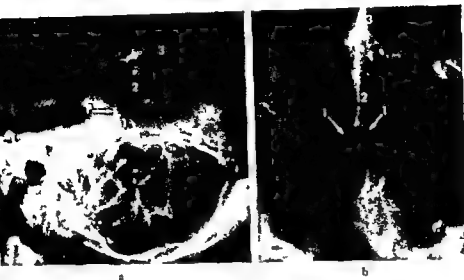


Fig 3 Normal precentral cerebellar vein. a) Venous phase of vertebral angiography, lateral view. The precentral vein (1, 2, 3) begins (1) in the precentral fissure between the lingula and the central lobule. The most anterior point (F) corresponds to the colliculo-central point. The vein runs upward a short distance in front of the cerebellum (2) and then above the cerebellum (3) in the cisterna ambiens as it enters the posterior end of the great vein of Galen. b) A half axial view. The two tributaries or brachia (1, 1) run medially and upward for a short distance before they fuse to form a single trunk (2) which continues upward and backward. The terminal portion of the vein (3) is obscured by the great vein of Galen and the straight sinus. The single midline trunk and the two diverging tributaries have the appearance of an inverted Y.

while the ponto-mesencephalic sulcus leads into it from in front. It should be emphasized that the structures in the anterior wall of the cleft—specifically the inferior colliculi, the brachia conjunctiva, the lingula and the anterior medullary velum—make up the roof of the lower part of the aqueduct and upper part of the fourth ventricle.

Because of the important anatomical relationships of the precentral fissure and its lateral extensions, a diagnostic landmark to determine the location and configuration of this cleft would be of considerable use. Occasionally on encephalography gas may enter this cleft. Under ordinary circumstances, however, no substantial amount of gas is seen below the level of the inferior colliculi. The precentral cerebellar vein and its tributaries described below are located in this cleft and appear to be more useful as landmarks for the structures in this region.

In a typical case (Fig 3) the precentral cerebellar vein is a single unpaired vessel in the midline. It runs upward in front of the anterior aspect of the superior vermis—that is, in front of the central lobule and the sulcus and

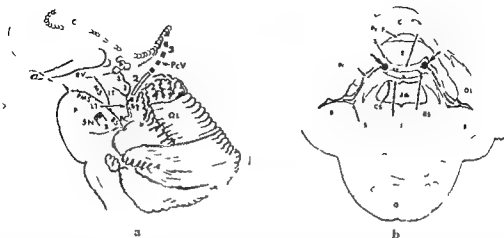


Fig. 2 a) Lateral view of posterior fossa structures with cerebellum in situ. Portions of superior vermis and adjacent hemisphere have been removed to expose brachium conjunctivum (BC) and brachium pontis (BI) as they join the cerebellum. The fine brachial vein (1) originates on brachium pontis (BI) and continues backward (a) over interbrachial sulcus (IBS) and brachium conjunctivum (BC) to lateral margin of lingula at lower end of paravermian sulcus. Small perforating veins (b) from the substance of the cerebellum join the vein in this region. The vein then runs upward and forward in precentral fissure (1) to reach the surface of cerebellum immediately behind inferior colliculi (1). A small lingular tributary (LT) from the lateral aspect of the lingula joins the vein at this level. Other labeled structures are Basal vein (BV), semispherical trigone (LTr), fifth nerve (V), quadrangular lobule (QL), lateral mesencephalic sulcus (LMS) and pontomesencephalic sulcus (PMS). b) Section through upper part of 4th ventricle. Plane of section is oblique to the long axis of the brain stem cutting through lower pons anteriorly and the brachium conjunctivum (BC) posteriorly. The mid portion (see 1 M) and the lateral extensions (see 1 L) of the precentral fissure are shaded. The two tributaries brachial veins (unlabeled arrows) which unite to form the precentral cerebellar vein proper are located on each side of the lingula (1). The dotted lines in the lateral extensions of the precentral fissure represent the brachial veins at levels below the plane of this section. The dotted line in the central portion of the fissure represents transverse communicating channels. Other labeled structures are Culmen (CU), central lobule (CL), preculminate fissure (PcuF), paravermian sulcus (PVS), wing of central lobule (AL), internal velo conjunctival sulcus (IVCS), external velo conjunctival sulcus (EVCs), anterior medullary velum (AMV), interbrachial sulcus (IBS), brachium pontis (BP), quadrangular lobule (QL).

is complicated by the numerous structures in this area. In the mid portion, these include from above downward, the medial portions of the inferior colliculi, the frenulum veli, and the lingula overlying the anterior medullary velum. The lateral extensions of the precentral cerebellar fissure extend not only laterally but also anteriorly and include in the anterior wall, the lateral portions of the inferior colliculi, the superior cerebellar peduncles and more laterally the lemniscal trigones and posterior aspects of the middle cerebellar peduncles. Between the superior cerebellar peduncle (brachium conjunctivum) and the middle cerebellar peduncle (brachium pontis) on each side, there is a sulcus designated as the interbrachial or interpeduncular sulcus (not the interpeduncular fossa). The anterior part of the wing rests in this sulcus. The lateral mesencephalic sulcus leads into the interbrachial sulcus from above,



Fig. 5 Normal variation of the precentral cerebellar vein. a) Half anterior-posterior view shows two independent vessels (2) which run upward and medially and unite to form a single trunk (3) in the upper part of the cisterna ambiens. These vessels begin as tortuous small veins (unlabeled arrows) located behind the midbrain on each side. b) Lateral view. The usual first portion of the precentral cerebellar vein is absent. Instead the vein appears to begin in a club-shaped density (anterior arrow) which represents the tortuous tributaries on the posterior aspect of the midbrain. The second portion of the vein (2) appears to be single but as indicated in the anterior projection is probably a double channel. A large superior vermian vein around the apex of the cerebellum runs forward to join the precentral cerebellar vein. As a result the third portion of this vessel (3) is unusually large. There is a sizeable anterior tributary of the superior vermian vein (posterior arrow). The lower portion of this vessel arises in the preculminate fissure while its upper part is directly applied to the anterior aspect of the culmen of the cerebellum.

precentral vein runs forward as well as upward to reach the surface of the cerebellum. At the point where the vein leaves the precentral fissure, the vessel lies immediately behind and below the inferior colliculi and makes a distinct turn backward as it continues upward in front of the superior vermis (culmen). The vein follows this upward course until it joins the posterior end of the great vein of Galen at its junction with the straight sinus.

The normal appearance on vertebral angiography of the precentral cerebellar vein and its tributaries corresponds to the anatomical relationships described above. In the lateral projection this vein appears to begin as a single trunk in the depths of the precentral cerebellar fissure. This portion of the vessel may be designated as the first portion. In some cases it appears to be composed in the depths of the fissure of superimposed tributaries of the two sides rather than a single trunk. However the point of union of these tributaries in the lateral projection is rarely evident. The course of the first

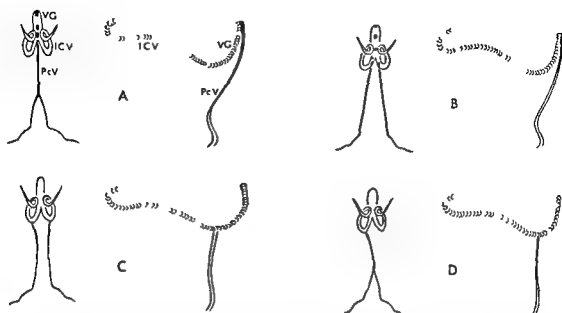


Fig. 1. Normal variations in the course of the precentral cerebellar vein and its tributaries. A) The two tributaries which arise over the brachium pontis and run medially extend upward for a variable distance as independent vessels before they fuse to form a single midline trunk. Precentral vein (PcV), internal cerebral vein (ICV), vein of Calen (VC). B) The point of union occurs unusually high or a short distance before the main trunk joins the posterior end of the great vein of Calen. As a result the usual single precentral cerebellar vein is represented by two rather long vessels for most of its extent. C) The precentral cerebellar veins are shown as completely independent vessels running a short distance from the midline and entering the internal cerebral vein of each side rather than the great vein of Calen. D) Occasionally the tributaries of the precentral cerebellar vein join to form a single trunk which runs upward to one side of the midline to enter the internal cerebral vein of that side.

drains into the posterior end of the great vein of Galen. It is formed by the union of symmetrical tributaries from each side which originate in the lateral extensions of the precentral cerebellar fissure and run medially within this fissure to join one another in the midline. Each tributary may be said to originate from small veins on the superior aspect of the brachium pontis and to run as a 'brachial vein' medially and backward across the interbrachial sulcus and the brachium conjunctivum to reach the posterior aspect of the lingula. As it runs over these structures, it assumes a configuration corresponding to their external contour. It is of interest that anatomical dissections show minute perforating twigs from the white matter of the cerebellum joining each brachial vein at the lateral margin of the base of the lingula or the lower end of the paramedian sulcus. Other fine veins from the lingula, colliculi and superior surface of the cerebellum may also join this venous pathway.

The union of the two symmetrical tributaries or brachial veins which form the single unpaired trunk, i.e. the precentral cerebellar vein proper, usually occurs in the depths of the precentral fissure. As a result, the first portion of the



Fig. 7 Venous phase of vertebral angiography, half axial ap view. The two brachial veins (a) are united by a transverse communicating branch (c). The combination of these vessels corresponds to the trapezoid outline of the roof of the upper portion of the fourth ventricle. The brachial veins are separated from the ventricle by the basilar conjunctiva and the communicating vessels by the lingula and the superior medullary velum. The superior continuation to and the midline of both brachial veins can be faintly seen (11).

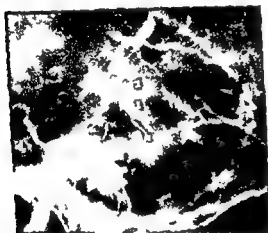


Fig. 8 Variation in the drainage of the precentral cerebellar vein, lateral view. The first (1) and second (2) portions of the precentral vein are small channels but normally situated. Drainage however is not only upward but also downward and forward via the brachial vein (a and a'). This vessel empties into the superior petrosal sinus via the petrosal vein.

which upward to reach the midline. These tributaries and the central unpaired stem give the appearance of an inverted Y with a long stem. Unfortunately in this projection the terminal portion of the vein is usually obscured by the overlying great vein of Galen and straight sinus.

The description given above of the typical precentral cerebellar vein and its main tributaries should be considered as a prototype subject to variations. Fig. 4. The point of union of the two tributaries may not occur exactly in the midline but rather a short distance to one side. More commonly the two tributaries fail to unite in the precentral cerebellar fissure itself but continue as individual trunks upward for a variable distance before joining (Figs 5 and 6). In such cases the two tributaries often communicate with each other

Fig. 6 Venous phase of vertebral angiography. Lateral view. The precentral vein (long transverse arrows) is double throughout much of its extent. A portion of the brachial vein (1) running over the brachium conjunctivum can be identified. This channel joins the main tributary in the precentral fissure almost at a right angle (short transverse arrow). This junction may be used to identify the lowermost point of the paravermian sulcus.



portion of this vein as it runs forward and upward is exactly parallel to the roof of the upper part of the fourth ventricle but separated from it by the thickness of the roof, or 2 to 4 mm. As the vein leaves the precentral fissure and turns backward between the central lobule of the cerebellum and the inferior colliculi, a characteristic obtuse angle or knee in the course of the vessel is created. The most interior point of this angle may be referred to as the 'colliculo central point', or for brevity, the 'CC point'. Similarly, the angle may be designated as the 'colliculo central angle' or simply as the 'CC angle'. For purposes of description, the next portion of the precentral cerebellar vein which continues upward and backward in front of the superior vermis may be designated as the second portion. It usually shows a slight curvature convex anteriorly. This portion of the vein does not run directly on the surface of the cerebellum but a short distance in front of the cerebellum. The distance between this vein and the surface of the cerebellum is well seen when the anterior tributary of the superior vermicular vein is also filled with contrast medium. The portion of the precentral vein above the level of the cerebellum within the central portion of the cisterna ambiens, that is the third or terminal portion of the vein, continues without interruption the upward and backward course of the second portion. This third part often shows slight convexity posteriorly although it may be straight or show a slight anterior convexity. The second and third portions of the vessel may therefore have a somewhat sinuous appearance or show a smooth anterior convexity. In the fronto occipital projection with the central ray tilted 25 to 30 degrees toward the feet (half axial *ap* view) the symmetrical tributaries or brachial veins which unite to form the unpaired precentral cerebellar vein run medially and some



Fig 10 Venous phase of vertebral angiography, lateral view. The tortuous portion of the precentral cerebellar vein (1, 2) as well as the colliculo-central point (P) are well seen. A somewhat tortuous cortical vein (unlabeled arrow) from the superior aspect of the cerebellar hemisphere runs medially upward and forward to join the precentral vein.



Fig 11 Normal variation of the precentral cerebellar vein. The first portion of the precentral cerebellar vein (1) and the colliculo-central point (P) have a normal configuration and location. However, the vessel extends vertically upward and enters the internal cerebellar vein (unlabeled arrow) rather than the posterior end of the great vein of Galen.

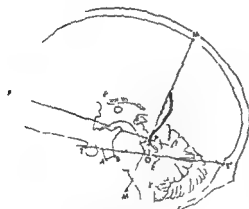


Fig 12 Normal measurements of the colliculo-central point. A variety of measurements from the colliculo-central point (P) to other anatomical landmarks are indicated. PA = the minimum distance from the chiasm to the pons. F = the inner table of the frontal bone. M = the anterior margin of the foramen magnum or the lower end of the clivus. V = the inner table of the parietal bone. T = the tuberculum sellae. T₁ = the tortula. TT = Twining's line. Q is the foot of a perpendicular line dropped from P to TT.

region of the flocculus to join the petrosal vein (Fig 8). More commonly, however, the midline precentral cerebellar vein is replaced by a somewhat similar vessel located in the lateral extension or wing of the precentral cerebellar fissure (Fig 9). This vessel runs upward and backward to one side of the midline but may also join the posterior end of the great vein of Galen. In the antero-posterior projection such a paracentral or lateral precentral cerebellar

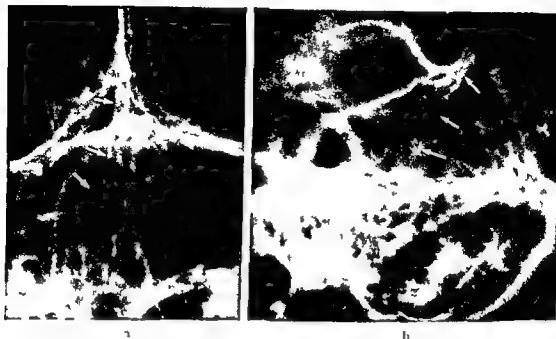


Fig 9 Piracentral or lateral precentral cerebellar vein. a) Half axial r.p. view. There is a sizeable rather long vein (arrows) which begins inferiorly at some distance from the midline and runs upward and medially in the lateral and central portions of the precentral cerebellar fissure to the paravermis sulcus. This vessel approaches but does not reach the midline until the upper portion of the cisterna ambiens. b) Lateral view showing the same vein (arrows) which resembles the usual midline precentral cerebellar vein. However, the first portion of the precentral vein and the knee or colliculo central angle are absent and this vessel is considerably straighter. As indicated in the r.p. projection, the portion of this vein in front of the cerebellum does not run in the midline but is more laterally located.

at various levels but particularly in the bottom of the precentral fissure by transverse connections designated as communicating veins (Fig 7). These anastomotic twigs are of special interest since they lie immediately behind the roof of the upper part of the fourth ventricle and, in combination with the brachial veins, serve to delineate the trapezoid configuration of the roof of the upper part of the fourth ventricle. There are numerous variations in the confluence of the precentral, superior vermian, internal occipital and medial temporal cortical, choroid, basal and other veins with the internal cerebral veins and the great vein of Galen. A not uncommon variation is the union of the precentral cerebellar vein with the superior vermian vein to form a short common stem within the cisterna ambiens. These variations in terminal drainage are of little diagnostic importance.

The precentral cerebellar vein is not evident in all patients. In some cases it may be quite small. In such instances, venous drainage may occur in an anterior direction via brachial veins which run forward and laterally to the

angulation in its course. In the lateral projection confusion may also arise when a cerebellar cortical vein on the superior aspect of the hemisphere runs upward and forward and may simulate the first portion of the precentral vein (Fig 10). Such a vessel however is located more posteriorly and does not show the typical colliculo-central angle. It should also be mentioned that the superior vermician vein usually has an anterior tributary which may be confused with the precentral vein. This tributary typically arises in the fissure between the culmen and the central lobule (preculminate fissure), runs directly on the anterior aspect of the culmen and therefore has a somewhat scalloped appearance. However the precentral cerebellar vein may occasionally be more posteriorly located than usually seen and replace the anterior tributary of the superior vermician vein. The precentral vein may also run unusually far forward and extend almost directly upward to join the internal cerebral vein to one side of the mid line (Fig 11).

As described above the first segment of the precentral cerebellar vein runs parallel and posterior to the roof of the upper part of the fourth ventricle and the lower portion of the aqueduct. Deformities and displacements of this segment and of the C-C point can therefore be interpreted in a fashion similar to the well known changes in the aqueduct and fourth ventricle seen on encephalograms. Measurements of the C-C point in relation to osseous anatomical landmarks may therefore be as useful as measurements of the aqueduct and fourth ventricle in encephalograms (Fig 12 Table). The minimal distance from this point to the posterior aspect of the clivus averaged 3.9 cm with a range of 3.6 to 4.3 cm as measured in 40 presumably normal adult angiograms. The fact that the perpendicular line dropped from this point to Twining's tuberculum torcular line approximately bisects the latter line is also a useful guide. In most cases with displacement or deformity of the precentral cerebellar vein visual inspection is sufficient to recognize the presence of an abnormality (Figs 13 and 14).

SUMMARY

The course and appearance of the precentral cerebellar vein is described in detail. The first portion of this vein within the precentral cerebellar fissure lies parallel to and behind the roof of the upper part of the fourth ventricle. The vein can be divided into three portions with the junction between the first and second portions forming a characteristic angle referred to as the colliculo-central angle. The most anterior part of this angle may be designated the colliculo-central point. The location of this point and the configuration of this angle is of diagnostic importance in recognizing deformities and displacements.



Fig. 13 Chordoma. Vertebral angiography, venous phase, lateral projection. A large calcified mass is present in the region of the destroyed dorsum sellae. The first and second portions of the precentral vein (1, 2) are displaced markedly backward. This is evident by the increased distance between this vessel and anterior structures such as the sella turcica and the clivus as well as the decreased distance to posterior structures such as the straight sinus. The colliculo-central angle (P) is flattened. The third portion of the vein (3) and adjacent great vein of Cullen are vertical in position.



Fig. 14 Tentorial notch meningioma arising from the junction of falx and tentorium. A large mass supplied by meningeal vessels (MM) is present in the region of the incisura. All three portions of the precentral cerebellar vein (1, 2, 3) are displaced forward and follow a single continuous curved course. The aqueduct and posterior portion of the third ventricle must also be displaced markedly forward in an arcuate fashion. Large choroidal veins (unlabeled arrow) within the velum interpositum are also evident.

vein is easily distinguished from the more common mid line precentral cerebellar vein. In the lateral view, however, these vessels may be confused. In this projection, the paracentral cerebellar vein is located more anteriorly than the usual precentral cerebellar vein and does not show any knee or

Table

Measurements of the colliculo central point — For measurement points see Fig. 17

	Lower limit	Upper limit	Mean		Lower limit	Upper limit	Mean
AP	3.6 cm	4.3 cm	3.9 cm	$\frac{TQ}{II'}$	44	55	49
BP	2.9 cm	3.6 cm	3.2 cm	$\frac{IP}{II'}$	60	72	66
MP	4.0 cm	5.8 cm	4.8 cm	$\frac{MP}{MM'}$	30	42	36%
QI	0.6 cm	1.6 cm	1.1 cm				

angulation in its course. In the lateral projection confusion may also arise when a cerebellar cortical vein on the superior aspect of the hemisphere runs upward and forward and may simulate the first portion of the precentral vein (Fig 10). Such a vessel however, is located more posteriorly and does not show the typical colliculo-central angle. It should also be mentioned that the superior vermian vein usually has an anterior tributary which may be confused with the precentral vein. This tributary typically arises in the fissure between the culmen and the central lobule (preculminate fissure) runs directly on the anterior aspect of the culmen and therefore has a somewhat scalloped appearance. However, the precentral cerebellar vein may occasionally be more posteriorly located than usually seen and replace the anterior tributary of the superior vermian vein. The precentral vein may also run unusually far forward and extend almost directly upward to join the internal cerebral vein to one side of the mid line (Fig 11).

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SUMMARY

The course and appearance of the precentral cerebellar vein is described in detail. The first portion of this vein within the precentral cerebellar fissure lies parallel to and behind the roof of the upper part of the fourth ventricle. The vein can be divided into three portions with the junction between the first and second portions forming a characteristic angle referred to as the colliculo-central angle. The most anterior part of this angle may be designated the colliculo-central point. The location of this point and the configuration of this angle is of diagnostic importance in recognizing deformities and displacements.

ZUSAMMENFASSUNG

Es wird der Verlauf und das Aussehen der präzentralen cerebellaren Vene im Detail beschrieben. Der erste Abschnitt dieser Vene in der präzentralen cerebellaren Fissur ist hinter dem Dach des oberen Teiles des vierten Ventrikels und parallel zu ihm gelegen. Die Vene kann in drei Abschnitte eingeteilt werden, wobei der Übergang vom ersten zum zweiten Abschnitt einen charakteristischen Winkel bildet, welcher als 'colliculo zentraler Winkel' beschrieben wird. Der vorderst gelegene Teil dieses Winkels mag als 'colliculo zentraler Punkt' bezeichnet werden. Die Lage dieses Punktes und die Form des Winkels ist von diagnostischer Bedeutung, in der Erkennung von Deformationen und Dislokationen.

RÉSUMÉ

Les auteurs décrivent en détail le trajet et l'aspect de la veine cérébelleuse précentrale. La première partie de cette veine dans le sillon cérébelleux précentral est située parallèlement au toit de la partie supérieure du quatrième ventricule en arrière de ce toit. On peut diviser la veine en trois parties. L'union de la première et de la seconde formant un angle caractéristique appelé 'angle colliculo central'. La partie la plus antérieure de cet angle peut être appelée 'le point colliculo central'. La localisation de ce point et la configuration de cet angle ont une importance diagnostique pour reconnaître les déformations et les déplacements.

REFERENCES

1. BROWNING W. The veins of the brain and its envelopes. Their anatomy and bearing on the intracranial circulation. I. B. O'Connor Jr. Brooklyn New York 1884.
2. BAILLY P. Intracranial tumors. 2nd ed. Charles C. Thomas Publishers. Springfield Illinois 1948.
3. DURR H. Recherches anatomiques sur la circulation de l'encephale. Arch. Physiol. norm. et path. 6 (1874) 316.
4. GREITZ T. and LINDEREN F. Cerebral vascular anatomy. In Angiography Vol. 1. Ed. by H. Abrams. Little Brown & Co. Boston 1961.
5. HAUGI I. Catheter vertebral angiography. Acta radiol. Suppl. 109 1954.
6. HIDEUX C. F. Etude anatomique sur la circulation veineuse de l'encephale. Thèse Bordeaux 1888.
7. HORMAN M. Zur vergleichenden Anatomie der Gehirn- u. Rückenmarksvenen der Vertebraten. Ztsch. f. Morphol. u. Anthropol. 3 (1901) 239.
8. JOHANSON C. Central veins and deep dural sinuses of brain. Anatomical and angiographic study. Acta radiol. Suppl. 107 1954.
9. MITTLER F. Neuroanatomy. The C. V. Mosby Co. St. Louis 1948.
10. PADGETT D. H. Development of cranial venous system in man from viewpoint of comparative anatomy. Contrib. Embryol. 36 (1957) 79.
11. POIRIER I., CHAMPY A. and NICHOLAS A. Traité d'anatomie humaine. Vol. III. Masson & Cie. Paris 1921.

PERCUTANEOUS VERTEBRAL ANGIOGRAPHY

by

H. KRAYENBUHL and M. G. YACARGIL

Within a ten year period 1 035 vertebral angiographies were done on 900 patients using the technique of percutaneous (ventro cervical) puncture of the vertebral artery. This procedure was carried out under general anesthesia exclusively in order to decrease the danger of arterial spasm as well as to render the puncture painless.

Good filling of the vertebral artery and its intracranial branches was obtained in 80 % of the cases. The contrast filling of the vessels was insufficient in 10 %, and in another 10 %, the puncture was unsuccessful. Complications were rare and minor; they consisted in either local or radicular pain both disappearing within a short time. One patient was lost owing to accidental intrathecal injection of the contrast substance. This fatal complication could have been avoided if proper attention had been given to the position of the needle.

The indication for the performance of angiography was given in the following groups of neurological disorders: Vascular diseases 365 cases (40.5 %), tumors 212 cases (23.1 %), cerebello-bulbo pontine syndrome (tumors not included) 275 cases and miscellaneous diseases (migraine, trauma, monoplegia, epilepsy and others) 66 cases (together 36.0 %).

ZUSAMMENFASSUNG

Es wird der Verlauf und das Aussehen der präzentralen cerebellaren Vene im Detail beschrieben. Der erste Abschnitt dieser Vene in der präzentralen cerebellaren Fissur ist hinter dem Dach des oberen Teiles des vierten Ventrikels und parallel zu ihm gelegen. Die Vene kann in drei Abschnitte eingeteilt werden, wobei der Übergang vom ersten zum zweiten Abschnitt einen charakteristischen Winkel bildet, welcher als colliculo zentraler Winkel beschrieben wird. Der vorderst gelegene Teil dieses Winkels mag als colliculo zentraler Punkt bezeichnet werden. Die Lage dieses Punktes und die Form des Winkels ist von diagnostischer Bedeutung in der Erkennung von Deformationen und Dislokationen.

RÉSUMÉ

Les auteurs décrivent en détail le trajet et l'aspect de la veine cérébelleuse précentrale. La première partie de cette veine dans le sillon cérébelleux précentral est située parallèlement au toit de la partie supérieure du quatrième ventricule en arrière de ce toit. On peut diviser la veine en trois parties. L'union de la première et de la seconde formant un angle caractéristique appelé angle colliculo central. La partie la plus antérieure de cet angle peut être appelée le point colliculo central. La localisation de ce point et la configuration de cet angle ont une importance diagnostique pour reconnaître les déformations et les déplacements.

REFERENCES

1. BROWNING W. The veins of the brain and its envelopes. Their anatomy and bearing on the intracranial circulation. F. B. O'Connor Jr. Brooklyn New York 1884.
2. BAILEY P. Intracranial tumors. 2nd ed. Charles C. Thomas Publishers. Springfield Illinois 1948.
3. DURFT H. Recherches anatomiques sur la circulation de l'encephale. Arch. Physiol. norm. et path. 6 (1874) 316.
4. GRFITZ T. and LINDERFEN F. Cerebral vascular anatomy. In Angiography Vol. 1. Ed. by H. Abrams. Little Brown & Co. Boston 1961.
5. HAUGI F. Catheter vertebral angiography. Acta radiol. Suppl. 109 1954.
6. HEDON C. E. Etude anatomique sur la circulation veineuse de l'encephale. Thèse Bordeaux 1888.
7. HOFMAN M. Zur vergleichenden Anatomie der Gehirn u. Rückenmarksvenen der Vertebraten. Ztsch. f. Morphol. u. Anthropol. 3 (1901) 239.
8. JOHANSON C. Central veins and deep dural sinuses of brain. Anatomical and angiographic study. Acta radiol. Suppl. 107 1954.
9. METTLER F. Neuroanatomy. The C. V. Mosby Co. St. Louis 1948.
10. PADGET D. H. Development of cranial venous system in man from viewpoint of comparative anatomy. Contrib. Embryol. 36 (1957) 70.
11. POIRIER P., CHARPY A. and NICHOLAS A. Traité d'anatomie humaine Vol. III. Masson & Cie. Paris 1921.

Table 2
Tumors (212 cases)

	No. of cases	Per cent
Supratentorial		
Frontal temporal parietal	14	6.6
Occipital	27 57	24.5
Mesodiencephal	25 57	24.5
Infratentorial		
Cerebello-pontine angle	36	
Cerebellum	77	
Pons	8 141	66.1
Cervical spine	3 5	2.4
Malformations	2 5	2.4

size vascularization and exact localization of details with respect to roentgenologic anatomy are sufficiently known (GALLOWAY & GREITZ 1960 WOLF et coll 1962 LASARGIL 1962 GREITZ & SJOGREN 1963 ECONOMOS & PROSA LENTIS 1963)

Vertebral angiography is of increasing diagnostic importance in cases with an unclear or atypical symptomatology with either isolated or combined signs of bulbar pontine or cerebellar lesions. Occasionally it is difficult to decide whether or not one is dealing with a vascular inflammatory degenerative posttraumatic or even neoplastic condition. It is of great value to exclude by means of a negative vertebral angiography the presence of a tumor. Thus an explorative occipital craniotomy may be avoided. In none of the 323 cases with negative angiograms was a tumor diagnosed even after a follow up of many years.

Vertebral angiography is a valuable aid in the diagnostic evaluation of certain atypical cases by permitting differentiation between a tumor and a vasculopathy such as an aneurysm or an angioma.

SUMMARY

Within a 10 year period (1953—1963) 1 035 percutaneous vertebral angiographies were performed in 900 cases. 23% were tumors (1/3 supra 2/3 infratentorial), 40% were cases with vascular disease and 37% cases with signs of an unknown pontine bulbar-cerebellar symptomatology. This investigation demonstrated that vertebral angiography is of great clinical value for the diagnosis of suspected intracranial tumor and of vascular diseases.

Table 1

Vascular diseases (365 cases)

	No. of cases	Per cent
Arteriosclerotic occlusion	80	21.9
Aneurysms		
Internal carotid artery and its branches	35	
Posterior communicating artery	23	22.1
Vertebrobasilar artery	23	81
Arteriovenous angiomas		
Cerebral hemispheres	27	
Mesodiencephalon	14	
Infratentorial	12	
Nuchal	5	58
Subarachnoid hemorrhage	157	43.0
Miscellaneous (fistula, persisting arteries, hematomas)	19	5.1

Pre- and postoperative vertebral angiography is performed in cases of subarachnoid hemorrhage and in cases of vascular occlusions to study the site of the lesion and the collateral circulation (Table 1).

Other angiographic methods such as puncture or catheterization of subclavian, axillary, brachial or femoral arteries are used to study extracranial morphological changes of the cerebral arteries. Extracranial portions are clearly demonstrated by these techniques.

Percutaneous vertebral angiography is particularly suited for the detection of tumors of intracranial localization. Tentorium, mesodiencephalon, retrosellar and occipito-median regions (Lorenz 1958), (Table 2). While carotid angiography gives very unsatisfactory results in these cases, vertebral angiography may even allow the drawing of conclusions concerning the type of tumor present.

Vertebral angiography is not very conclusive in the presence of an infratentorial space-occupying lesion. Once the tumor has reached a certain size, the clinical symptomatology becomes very clear. An exploration being performed bilaterally in any case, further angiographic localization becomes unnecessary. A small tumor, on the other hand, would cause an insignificant displacement of the vessels. However, the fact must be recalled that a number of tumors often exhibit readily detectable pathological vessels (meningiomas, neurinomas, angiomas, metastases, plexus papillomas, glomus tumors). Angiography allows the diagnosis of these lesions concerning their number,

Table 2
Tumors (212 cases)

	No. of cases		Per cent
Supratentorial			
Frontal temporal parietal	14		6.6
Occipital	27	52	24.5
Mesodiencephalic	25	52	24.5
Infratentorial			
Cerebellopontine angle	56		
Cerebellum	77		
Pons	8	141	66.1
Cervical spine	3	5	2.4
Malformation	2	5	2.4

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Posterior communicating artery	23	
Vertebro-basilar artery	23	
Arteriovenous angiomas		
Cerebral hemispheres	27	16.0
Mesodiencephalon	11	
Infratentorial	12	
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Miscellaneous (fistula, persisting arteries, hematomas)	19	5.2

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NEURORADIOLOGISCHE BESONDERHEITEN IM FRÜHSTADIUM DER GROSSHIRN TUMOREN UND BEI REZIDIVEN

von

W. KRENNEL

Von der Kontrastmittel Untersuchungen verlangen wir zunächst den Nachweis eines raumfordernden Prozesses überhaupt und zwar in einem möglichst frühen Stadium sodann für die Therapie eine genaue Aussage über die Lokalisation und Ausdehnung und schließlich wird auch ein Beitrag zur Artdiagnose erwartet.

Bei den Großhirn Tumoren wenden wir zuerst die Serien Angiographie an, da sie lokalisatorisch und vielfach artdiagnostisch von entscheidendem Wert ist. Im Hinblick auf die Frühdiagnose sind jedoch die Grenzen der Aussage-möglichkeit zu berücksichtigen. Dies gilt besonders für die häufig nicht durch pathologische Gefäße dargestellten langsam wachsenden Gliome.

Der Bericht stützt die neuroradiologischen Befunde bei 465 histologisch verifizierten Großhirn Geschwulsten ohne Stauungspapille und 100 Rezidiv Tumoren. Die Diagnose des Tumors basierte in 60 % auf dem angiographischen Befund, in 6 % war allein eine Encephalographie angefertigt worden. Bei

ZUSAMMENFASSUNG

Innerhalb der letzten 10 Jahre (1953—63) sind an 900 Patienten 1 035 perkutane vertebrale Angiographien durchgeführt worden, 23 % waren Tumoren (1/3 supra 2/3 infratentoriell) 10 % vasculäre Erkrankungen 37 % mit Anzeichen einer unbekannten pontin bulbären cerebellären Symptomatologie. Die Untersuchung zeigte, dass die vertebrale Angiographie von grosser klinischer Bedeutung für die Diagnosestellung intrakranieller Tumoren und vaskulärer Erkrankungen ist.

RÉSUMÉ

Au cours d'une période de 10 ans (1953—1963) les auteurs ont fait 1 035 angiographies vertébrales percutanées sur 900 sujets. 23 % avaient des tumeurs (1/3 supra 2/3 infratentorielles) 40 % une affection vasculaire et 37 % présentaient une symptomatologie pontobulbo-cérébelleuse de cause inconnue. Ce travail montre que l'angiographie vertébrale a un grand intérêt clinique pour le diagnostic de tumeur intracrânienne et d'affection vasculaire.

REFERENCES

- ECONOMOS D. et PROSMANTIS A. L'artère cérébelleuse supérieure dans les tumeurs de la fosse postérieure. *Acta radiol.* 1 (1963) 207.
- HAUCE T. The catheter vertebral angiography. *Acta radiol. Suppl.* 109 (1954).
- GALLOWAY J. R. and GREITZ T. The medial and lateral choroid arteries. *Acta radiol.* 53 (1960) 353.
- GREITZ T. and SJÖGREN S. E. The posterior inferior cerebellar artery. *Acta radiol.* 1 (1963) 284.
- KRAYENBUHL H. und YAŞARGIL M. G. Die vaskulären Erkrankungen im Gebiet der Arteria vertebralis und Arteria basilaris. G. Thieme, Stuttgart 1957.
- LOFFREN I. O. Vertebral angiography in the diagnosis of tumors in the pineal region. *Acta radiol.* 50 (1958) 108.
- NAMIN P. L'artériographie vertébrale. G. Doin, Paris 1955.
- OLSSON O. Vertebral angiography in the diagnosis of acoustic nerve tumors. *Acta radiol.* 39 (1953) 265.
- RADNER S. Vertebral angiography by catheterization. *Acta radiol. Suppl.* 87 (1951).
- SJÖGREN S. E. Percutaneous vertebral angiography. *Acta radiol.* 40 (1953) 113.
- WOLF B. S., NEWMAN C. M. and KIHINANI M. T. The posterior inferior cerebellar artery on vertebral angiography. *Amer. J. Roentgenol.* 87 (1962) 322.
- YAŞARGIL M. G. Die Vertebroisangiographie. Ihre Bedeutung für die Diagnose der Tumoren. *Acta neurochir. Suppl.* 9 (1962).

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33 % erforderte die Angiographie zur genauen Lokalisation eine Ergänzung durch eine Enzephalographie

Tabelle 1

Diagnose von 465 Großhirntumoren ohne Stauungspapille

Röntgen Nativbild allein	1 °
Carotis Angiographie	60 °
Enzephalographie	6 °
Carotis Angiographie und Enzephalographie	33

Bei einer früheren Untersuchung, die alle Großhirn Geschwulste unabhängig vom Grad der intrakraniellen Drucksteigerung einbezogen hatte, ergab sich nur in 17,5 % die Notwendigkeit einer kombinierten Kontrastmitteluntersuchung. Das heißt, im Frühstadium des Geschwulstwachstums darf man sich

Tabelle 2

Angiographische Befunde bei Crosshirn Tumoren ohne Stauungspapille

	Meningiome	Astrocyt	Oligod	Sonstige Gliome	Chorblast	And mal Tu	-
	110	99	96	43	89	28	465
Angio	89	77	77	28	83	23	377
Normal	3	3	,	3	2	2	18
Irregl Bfld	4	5	12	2	2	—	25
Apericall Verl	2	6	9	1	7	1	26
Arter Gefäß V	20	40	31	15	50	7	143
Venen Ausfall	4	8	3	2	11	2	27
Pathol Gefäße	55	15	16	5	33	10	134
Nicht bewert	1	—	1	—	1	1	4
EG	47	63	58	20	19	8	215
VC	5	13	13	7	8	2	48
Normal	—	1	1	1	3	1	7
Balken Septum							
Schragstand	1	3	6	1	1	—	12
Seiten Verlag	4	13	—	1	2	—	20
		2			3		5
Positiv Bfld	36	40	39	15	11	7	148
„	5	10	13	6	4	2	40
Tu Seite Ø Fllg	11	2	3	2	1	—	10
		1					2
Ø Fllg	4	4	11	—	1	—	18
					1		1

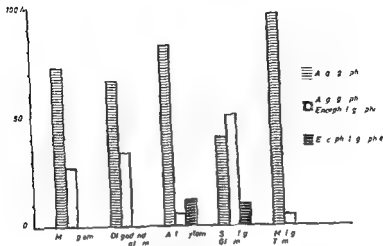


Abb. 1 Anteil der ein Kontrastmittel Verfahren bei der Diagnose der cerebralen Tumoren (100 Tumoren)

bei einem klinisch begründeten Tumorverdacht nicht mit einer negativ ausgefallenen Kontrastmittel Methode begnügen

Betrachten wir die bei den verschiedenen Tumorarten erzielten Ergebnisse (Tab. 2). Es fällt der große Anteil normaler oder fraglicher angiographischer Befunde bei den Oligodendrogliomen (in 22 %) aber auch Ependyomen und Spongioblastomen (in 18 %) auf. Die Zahl normaler Encephalogramme ist absolut und relativ geringer. Die bei den Astrozytomen recht oft beobachtete isolierte Seitenverlagerung des Ventrikel Systems ist auf die sich diffus ausbreitenden Tumoren zu beziehen. Bei den Glioblastomen mit normalen Ventrikelbildern handelte es sich um ein Frühstadium der Tumor Entwicklung.

Tabelle 3

Angiographische Befunde bei frontalen und parietalen Gliomen je Malignitätsgrad

	Angiographie				Encephalographie		
	Normal	Fragl. Befund	Alle 1 A. pericall. Verl.	Positiver Befund	Normal	Fragl. Befund	Positiver Befund
Lang wachsende Gliome	5	11	3	16	3	5	20
Maligne Gliome = Astrocytome	—	4	1	21	—	—	18

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	Meningiome	Astrocyt	Oligod	Sonstige Chome	Glioblast	And mal Tu	
	110	99	96	43	89	78	465
Angio	89	77	77	28	83	23	377
Normal	3	3	5	3	2	2	18
Irregl Bfld	4	5	17	2	2	—	25
Arter. Gefäß Verl	2	6	9	1	7	1	26
Arter. Gefäß V	20	40	31	15	30	7	143
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Schrägstand	1	3	6	1	1	—	12
Seiten Verlag	4	13	—	1	2	—	20
		2			3		5
Positiv Bfld	36	40	39	15	11	7	148
" "	5	10	13	6	4	2	40
Tu Seite Ø Illg	2	2	3	2	1	—	10
" "		1					2
Ø Illg	4	4	9	—	1	—	18
					1		1

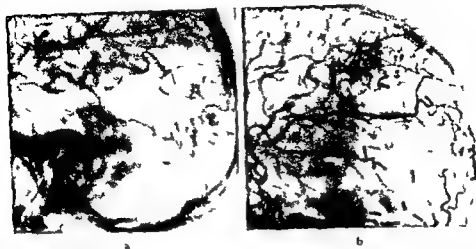


Abb 3 Occipitales Glioblastom a) Carotis-Angiographie präoperativ Tumorgefäße occipital
b) Carotis-Angiographie 1 1/2 Jahre postoperativ Neue Tumorgefäße parietal (Rezidiv)

was das Neoplasma im Angiogramm sowohl durch die Parallelverschiebung der A. pericallosa als auch durch eine Darstellung pathologischer Gefäße zu erkennen

Bei Berücksichtigung der Tumorlokalisation hat die Angiographie vor allem bei den parasagittalen, den in den Stammganglien gelegenen und den parieto-occipitalen Prozessen die meisten Versager. Der Unterschied zwischen dem angiographischen und encephalographischen Befund kann sehr erheblich sein. Bei parasagittalen Geschwulsten sind Röntgenaufnahmen im Sitzen zur frühzeitigen Erfassung der Glome unerlässlich.

Neben der unterschiedlichen Aussagemöglichkeit der beiden Kontrastverfahren je Tumorart und Lokalisation kommt auch dem Ausmaß der Umgebungsreaktion große Bedeutung zu. Unterscheidet man bei den Oligodendrogliomen und Astrozytomen zwischen den langsam und rasch wachsenden, so ergibt sich eindeutig, daß bei der ersten Gruppe die Zahl der unsicheren Befunde größer ist. Andererseits versagte bei den (rasch wachsenden) Gliomen II Grades das Encephalogramm in keinem Fall (Tab. 3). Die langsam wachsenden Glome, die klinisch fast ausnahmslos unter dem Bild eines Anfalls leidens in Erscheinung treten, stellen die schwer diagnostizierbare Tumorgruppe dar. Dazu kommt, daß auch die Isotopenuntersuchung in diesen Fällen in 40% negative Ergebnisse liefert. Ebensovienig ist von der Echoencephalographie in diesem Stadium zu erwarten.

Unter unseren 100 Rezidiv-Tumoren über deren Kontrastmittel Befunde

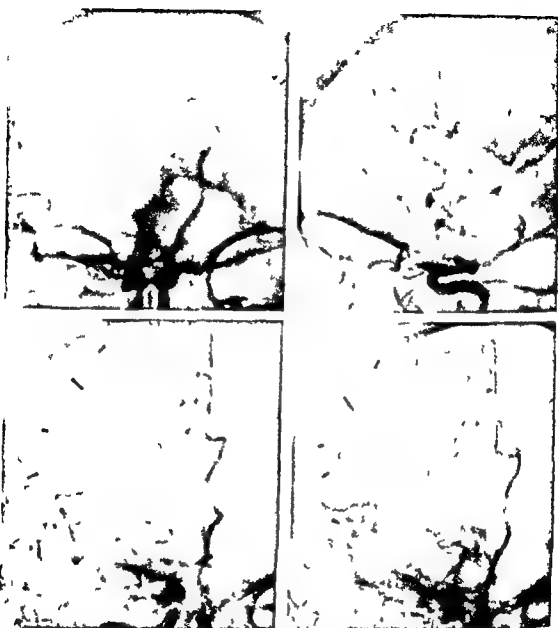


Abb 2 Präzentrales Glioblastom. *Oben links*: Carotis-Angiographie präoperativ (v.p.) Bogenförmige Verlagerung der A. pericallosa. *Oben rechts*: Seitlich — Lithologische Gefäße präzentral. *Unten links*: Carotis-Angiographie (4 Monate postoperativ). Nur noch geringe Verlagerung der A. pericallosa. *Unten rechts*: Carotis-Angiographie (10 Monate postoperativ). Erneute Zunahme der Massenverschiebung (Rezidiv).

In dem Fall einer 49-jährigen Pat. mit einem Glioblastom ergab sich zunächst weder im Angiogramm noch im Encephalogramm ein eindeutiger Hinweis auf einen raumfordernden Prozeß. Zwei Wochen später konnte lediglich ein Schragstand der Balkenzisterne registriert werden. Nach weiteren 6 Wochen



Abb 3 Occipitalen Glioblastom a) Carotis-Angiographie präoperativ b) Carotis-Angiographie 5 Jahre postoperativ

was das Neoplasma im Angiogramm sowohl durch die Parallelverschiebung der Arterien als auch durch eine Darstellung pathologischer Gefäße zu erkennen

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Unter unseren 100 Rezidiv-Tumoren lieferten die Kontrollmittel Befunde

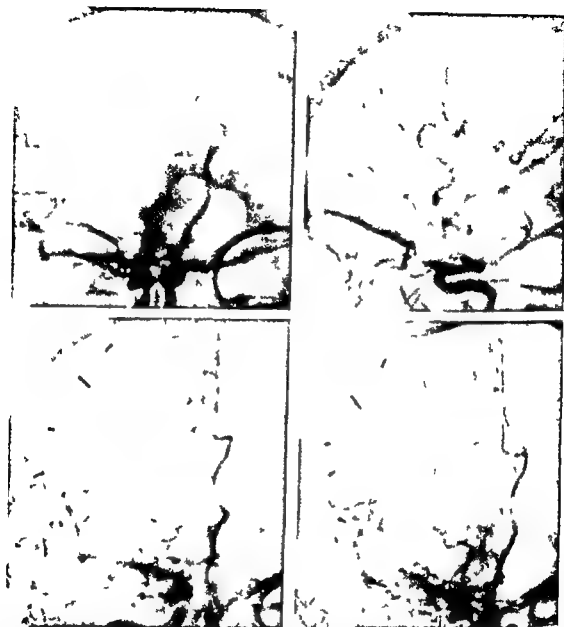


Abb. 2 Perizentrales Glioblastom. Oben links: Carotis-Angiographie präoperativ (a.p.) Bogenförmige Verlängerung der A. pericallosa. Oben rechts: Seitlich — Pathologische Gefäße präzentral. Unten links: Carotis-Angiographie (4 Monate postoperativ). Nur noch geringe Verlängerung der A. pericallosa. Unten rechts: Carotis-Angiographie (10 Monate postoperativ). Erneute Zunahme der Massenschiebung (Residiv).

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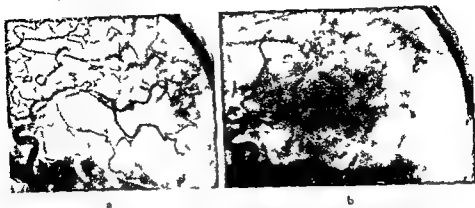


Abb 4 Temp Oligode drogl om a) Carot s Angiographie praoperativ Anhebung der A cerebri med gef ßf einer Raum b) Carotis-Angiographie 2 1/2 J postoperativ Tumorgefäße parietal temporal (Rez d v)

rechts) an der neben der Pericallosa die wieder zur Gegenseite verlagert ist auch die Media teilnimmt Diese ist in ihrem Anfangsteil bogig nach unten verlagert Das Rezidiv lag im vorderen Frontalmark ohne daß ■ zu der sonst typischen bogenförmigen Verlagerung der A pericallosa kam Betrachte wir nun den Primartumor so fällt gegenüber dem Rezidiv folgendes auf 1 Primar bestand der für einen frontalen Tumor typische Pericallosa verlauf 2 das Rezidiv fand sich an anderer Stelle es war von der Grenzzone der Resektion weitergewachsen und 3 nur praoperativ waren Tumorgefäße im Angiogramm nachweisbar Die frontalen Gliom Rezidive lassen fast stets den typischen Pericallosa Verlauf im antero posterioren Strahlengang vermissen Als Erklärung bieten sich die durch die Erstoperation gesetzten Veränderungen wie Hirngewebe Resektion und gliose Narbenbildung an

Der Ortswechsel des Rezidivs ist sehr häufig zu beobachten da sich dieses von der Grenzfläche der primären Resektion aus weiterentwickelt Auch das folgende Angiogramm von einem occipitalen Glioblastom (Abb 3 a und b) zeigt eine Wanderung des Tumors Außerdem kommt es in diesem Fall 1 1/2 Monate nach der Entfernung des ersten Tumors sekundär zur Ausbildung neuer pathologischer Gefäße

In beiden zuletzt demonstrierten Fällen war zusätzlich eine lokale Behandlung mit Kobalt 60 erfolgt Man konnte geneigt sein das Verschwinden der Tumorgefäße als eine Folge der Bestrahlung aufzufassen Jedoch zeigte eine Zusammenstellung aller postoperativ bestrahlten Fälle sei es mit Röntgenstrahlen mit dem Betatron oder Kobalt daß ein statistisch beweisbarer Einfluß auf die Tumorgefäße gegenüber den nicht bestrahlten nicht vorlag Im

Tabelle 4

*Einfluß der Röntgenbestrahlung oder ⁶⁰Co Behandlung auf die im Angiogramm nachweisbaren Tumor-
gefäße bei Oligodendrogliomen Astrozytomen Ependymomen*

Gesamt Zahl der Gliome	Primär Rezidiv	Tu Gefäße + Tu Gefäße Ø	Primär Rezidiv	Tu Gefäße Ø Tu Gefäße +	Primär = Rezidiv
Keine Röntgen oder ⁶⁰ Co Behandlung 31	2		5		24
Postoperative Röntgen oder ⁶⁰ Co Behandlung 27	1		13		13
58	3		18		37

hier berichtet werden soll, finden sich je 20 % Meningiome und maligne Tumoren, d. h. Glioblastome und Sarkome. Die restlichen 60 % werden durch die Gruppe der langsam wachsenden Gliome gestellt.

Die Bestätigung des Rezidivs allein durch die Angiographie gelingt am häufigsten bei den Glioblastomen und Sarkomen (Abb. 1). Dagegen war bei der Gruppe der langsam wachsenden Gliome in rund 30 % eine ergänzende Untersuchung durch eine Encephalographie erforderlich. Der Anteil ist bei den Ependymomen und Spongioblastomen ebenso wie bei der Diagnostik des Primärtumors im großen Ausmaß jedoch der Unterschied zwischen den Oligodendrogliomen und Astrozytomen. Diese Differenz erklärt sich aus der Tatsache, daß bei den Astrozytomen die Massenverschiebung in der Mehrzahl der Fälle auch beim Kontrollangiogramm zu finden ist, während bei den Oligodendrogliomen die Verlagerung der A. pericallosa sehr oft beim Rezidiv fehlt oder sogar zur operierten Seite hin erfolgt.

Auf den Wechsel der Massenverschiebung haben bereits LINDGREN u. Mitarbeiter bei den Untersuchungen über die Rezidiv Diagnostik mit Tantalum-Puder aufmerksam gemacht. In den ersten Wochen und Monaten bildet sich die Massenverschiebung zurück, und die A. pericallosa wird infolge der Resektionsbedingten Hirnatrophie auf die Operationsseite verlagert.

Bildet sich das Rezidiv im gleichen Hirnlappen, so kann nach anfänglicher Normalisierung im Gefäßverlauf eine erneute Verlagerung auftreten. Im folgenden Fall eines frontoprazentralen Glioblastoms (Abb. 2, oben links u. rechts) ist der Verlauf der A. pericallosa und der A. cerebri media 4 Monate postoperativ annähernd wieder normal (Abb. unten links). Nach weiteren 6 Monaten ist wieder eine Massenverschiebung zu erkennen (Abb. 2, unter

LITERATUR

- BREIT A und PREIFFER I Mehrfache Angiographien während der Röntgenbestrahlung von Hirngeschwulsten Acta radiol 46 (1956) 469
- FRANCESCONI G MARZANO E e PAGLIUCCI A Rilievi angiografici nei processi espansivi endocranici dopo intervento operatorio e trattamento radiante Gazz int Med Chir 64 (1959) 3250
- FRIEDMANN G KRENKEL W und TONNIS W Angiographie oder Pneumencephalographie? Fortschr Röntgenstr 96 (1962) 181
- GLONING K Tumorzidive im Angiogramm Acta neurochir Suppl 111 (1955) 250
- KRENKEL W und KROHN G Grenzen der Kontrastmitteldiagnostik im Frühstadium der Großhirntumoren 45 Tagg Dtsch Röntgengesellschaft, Fortschr Röntgenstr 1965 Teil A p 320
- LINDGREN M LOFGREN F O and LUNDBERG N Tantalum powder as an indicator of the brain tumour region for postoperative radiotherapy and the diagnosis of recurrence Acta radiol 48 (1957) 17

Gegenteil ergab sich sogar, daß die Rate der Kontrastmittelgefüllten Rezidive unter den postoperativ bestrahlten Gliomen größer war als bei den nicht bestrahlten Fällen (Tab. 1).

Bei den folgenden Pat. (Abb. 4a) wurde 1951 ein temporales Oligodendrogliom entfernt und eine Röntgen-Nachbestrahlung mit einer Herddosis 3 000 R durchgeführt. 2 1/2 Jahre später zeigte sich angiographisch (Abb. 4b) ein Rezidiv, das sich nunmehr durch zahlreiche arterio-venöse Fisteln, ähnlich einem Glioblastom, markierte.

Der Anteil der pathologischen Gefäßdarstellungen steigt bei den primär langsam wachsenden Gliomen von 15 % auf 40 % beim Rezidiv-Tumor an. Der Nachweis einer vorzeitigen Venenfüllung aus dem Tumorgebiet kann als sicherer Hinweis auf eine maligne Degeneration des Neoplasmas gelten. Außerdem sind Verlagerungen der inneren Venen zu berichten, die ein Rezidiv andeuten können, während eindeutige Verlagerungen der Hauptarterien noch nicht vorliegen.

ZUSAMMENFASSUNG

Im Frühstadium der Großhirn-Geschwülste bewährt sich die Angiographie sowohl in lokalisatorischer als auch artdiagnostischer Hinsicht. Die langsam wachsenden Gliome erfordern häufiger als in einem späteren Stadium des Tumorstadiums eine ergänzende Enzephalographie. Bei den Rezidiven sind die infolge der postoperativen Atrophie und Narbenbildung auftretenden Gefäßverlagerungen von den Veränderungen, die durch ein Rezidiv hervorgerufen sind, abzugrenzen. Verlagerungen der Arteria pericallosa zur Gegenseite bleiben beim Rezidiv oft aus. Der Anteil pathologischer Gefäßdarstellungen steigt bei den Gliomrezidiven erheblich an.

SUMMARY

Angiography is of proven merit in the localisation and establishment of the type of early cerebral tumours. Supplementary encephalography is more often required in the early stages of slow growing gliomas but infrequently in advanced cases. Recurrence necessitates vascular changes due to postoperative atrophy to be distinguished from those due to neoplastic scarring. Displacement of the arteria pericallosa is often absent in the case of recurrent neoplasms. The number of abnormal vessels is much increased in recurrent gliomas.

RÉSUMÉ

Au stade précoce des tumeurs du cerveau l'angiographie a fait ses preuves aussi bien pour la localisation que pour le diagnostic de nature. Les gliomes à croissance lente nécessitent plus souvent une encéphalographie complémentaire à leur stade de début qu'à un stade ultérieur. Dans les récidives il faut distinguer les déplacements de vaisseaux qui sont dus à l'atrophie et à la cicatrice de ceux qui sont causés par une récidive. Les déplacements de l'artère péricallosale vers le côté opposé sont fréquents dans les récidives. La proportion d'aspects vasculaires pathologiques augmente considérablement en cas de récidive de gliome.

LITERATUR

- BREIT A und PFEIFFER I Mehrfache Angiographien während der Röntgenbestrahlung von Hirngeschwulsten *Acta radiol* 46 (1956) 469
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POSSIBLE MECHANISMS OF COMPLICATIONS OF ANGIOGRAPHY

by

H. KUTT, K. VEREBILY, N. BANC I, STREUTH and I. McDOWELL

Angiographic procedures employing water-soluble iodinated contrast media are of great importance in clinical medicine. They are, however, associated with complications which range from mild subjective complaints to severe objective findings or death. The mild side effects are relatively frequent and have been reported to have an incidence of up to 33% (PENNINCRESS *et coll* 1955, FIELD *et coll* 1962, CULP *et coll* 1957). Severe complications, fortunately, are rare. Some contrast media have been found to be more toxic than others and the use of some earlier contrast media has been abandoned in favor of newer substances that have a lower incidence of complications.

The mechanisms of complications are not well understood. Some authors believe that all complications can be explained by mechanical factors such as faulty techniques or accidental dislodgement of a pre-existing atherosclerotic plaque or thrombus (SCHEINBLUM & ZUNIFER 1963). Others have found that contrast media have biological effects, which may in part be responsible for complications. Changes in capillary blood flow (SORBIN *et coll* 1959, JOHANSON & KNISLEY 1962), capillary fragility, and increased capillary permeability have

been reported (FOLTZ et coll 1952 BASSETT et coll 1953, BROMAN & OLSSON 1949) The cardiac and respiratory rate have been found to be altered (EPSTEIN et coll 1959 GREITZ & WEISS 1959) A direct toxic effect upon cells has been thought to be responsible for certain complications (VERASIMIDES et coll 1963)

Our own studies have been concerned with the biological effects of contrast media on blood and its constituents and the possible relationships of such effects to the occurrence of complications The intravascular concentrations of contrast media during and following the injections for cerebral angiography have been measured This information was necessary for the evaluation and design of meaningful *in vitro* and animal experiments for demonstration of the biological effects of contrast media upon blood constituents At concentrations commonly encountered during angiography, changes in blood proteins red cells electrolytes and coagulation were found We believe that these changes may contribute to the development of certain types of complications

Materials and methods

Contrast media included in these studies were sodium diatrizoate, methyl glucamine diatrizoate (generously supplied by Winthrop Laboratories) sodium iothalamate methylglucamine iothalamate, sodium acetrizate and iodopyracet

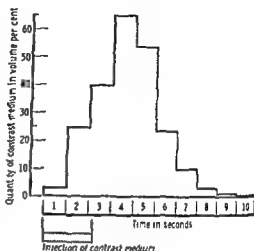
Patients included in these studies were individuals with a variety of *neurologic* disorders including many with cerebrovascular disease undergoing cerebral angiography for diagnostic purposes Also blood from young healthy volunteers was collected for controls of *in vitro* experiments

The intravascular concentrations of contrast media in the carotid artery during retrograde brachial angiography were measured with the help of a continuous sampling device previously reported from our laboratory The concentration of contrast medium in the carotid blood samples was determined with a phosphotungstic acid reagent or estimated from the dilution of Evans blue which had been previously mixed with the contrast media (KUTT et coll 1963)

Paper electrophoresis of blood proteins was carried out in Spenco Model R Durrum type paper electrophoresis cell with barbital buffers as previously reported (KUTT & McDOWELL 1962) The strips were scanned with a recording densitometer Starch gel electrophoresis was carried out according to the technique of SMITHIES (1955)

The combined concentrations of ionized calcium and magnesium were determined with EDTA titration according to the technique of COULSEN & HERNANDEZ (cf GRADWOL 1956)

Fig. 1 The dotted area represents quantities of contrast medium by volume in the common carotid area of a patient during retrograde brachial angiography. A peak of 65% by volume was reached in the 4th second after the start of injection. The injection of 30 ml 50% sodium diatrizoate lasted 2.2 seconds and is indicated by arrows.



Counting red cell clumps was carried out in a specially designed counting chamber utilizing the principles of Breed counting. The samples were prepared from 5 ml of venous blood collected into tubes containing EDTA as anticoagulant. Six parts of blood were added to 4 parts of contrast medium, mixed on a Vortex Jr Mixer for 5 sec and allowed to stand for 4 min and counted. All counts were carried out within 30 min after drawing the blood and averages of 5 counts were calculated.

The mesenteric circulation of albino male 300 gm rats of the Wistar strain previously anesthetized with 100 mg/kg of Nembutal given intramuscularly was studied. Through a microscope the mesentery was observed on a horizontal observation stage while contrast medium was injected into the proximal stump of the carotid artery as previously reported (Kutt et al. 1963). This technique was adapted from Zweirach and Metz (1956). Motion pictures of the mesenteric circulation were made before and after injection.

The clotting time was determined according to the Lee White method. A variety of components of the clotting system was determined according to the techniques of Owen (1947), Quick (1937) and Bachmann et al. (1957).

The electrical conductivity of contrast media was measured in a L&B type 5313 conductivity flow cell with a Model RC 16B2 Industrial Instruments conductivity bridge.

Results

Measurement of intravascular contrast medium concentrations during cerebral angiography in man. Intrarterial and intravenous concentrations of contrast medium during and immediately after the injection of contrast medium were measured in 10 patients undergoing right retrograde brachial angiography. Thirty ml

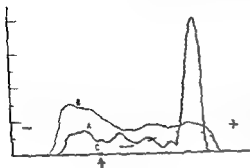


Fig 2 Densitometric recordings of paper electrophoresis patterns of normal serum protein (curve A) a mixture of 5 parts of serum and 5 parts of 50% sodium diatrizoate (curve B) and 50% sodium diatrizoate alone (Curve C)

contrast medium was injected into the innominate artery. Sampling from the right common carotid artery revealed in one patient that as much as 65% of its content for a few seconds consisted of contrast medium. Fig 1 shows that the peak concentration of contrast medium was reached in the 4th second after the injection was started. Then the concentration gradually fell to nearly zero within 9 seconds. Peak values of about 50% by volume of contrast medium were seen in 6 other patients. In 3 patients peak concentrations of 35 to 40% by volume were observed.

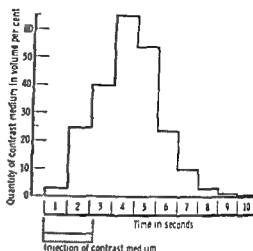
A concentration of contrast medium of 72% by volume for 1/2 second was found using a model which simulated the actual pressure and flow rate in the human carotid artery as might be present in a patient at the time of direct carotid angiography. This suggests that during direct carotid angiography higher peak concentrations of contrast medium might occur in cerebral blood than with brachial angiography.

Venous concentrations were measured in blood taken from the jugular bulb during and after the injection of 30 ml of contrast medium for retrograde brachial angiography. Peak concentrations of 9 to 16% by volume lasting 3 to 6 seconds were observed.

These experiments indicated that considerable displacement of blood in the cerebral vessels takes place and the mixing ratios of blood and contrast medium in the carotid artery can be as high as 6 parts of contrast medium and 4 parts of blood.

Changes of electrophoretic mobilities of blood proteins. Mixing plasma, serum, or whole blood with contrast media causes changes in the electrophoretic pattern of blood proteins. The normal albumin and alpha and beta globulin peaks flattened and a high peak developed in the gamma globulin area. These changes became more marked as the concentration of contrast media increased.

Fig. 1 The dotted area represents quantities of contrast medium by volume in the common carotid area of a patient during retrograde brachial angiography. A peak of 60% by volume was reached in the 4th second after the start of injection. The injection of 30 ml 50% sodium diatrizoate lasted 2.2 seconds and is indicated by arrows.



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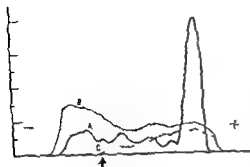


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Changes of electrophoretic mobilities of blood proteins. Mixing plasma serum or whole blood with contrast media causes changes in the electrophoretic pattern of blood proteins. The normal albumin and alpha and beta globulin peaks flattened and a high peak developed in the gamma globulin area. These changes became more marked as the concentration of contrast media increased.

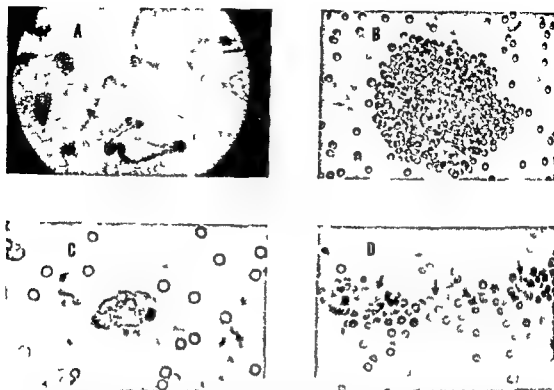


Fig 3 Photomicrographs of clumps formed in a mixture of 7 parts whole blood and 3 parts of 50 % sodium diatrizoate. A) Round, oval and strand like clumps. Magnification 8×25 . B) Round clump showing small clear area left from center. Magnification 11×40 . C) The clear center stained with bromphenol blue: most of the red cells have been removed by crushing: increased contrast over the unstained clear area in B. D) Clear strand stained with bromphenol blue joining 2 round clumps with clear centers marked with arrows: red cells partly removed by crushing.

Fig 2 shows densitometer recordings of 1) a normal protein pattern (Curve A), 2) a mixture of 5 parts of serum and 5 parts of 50 % sodium diatrizoate (Curve B), and 3) 50 % sodium diatrizoate alone (Curve C). As can be seen, some protein in the mixture moved with the contrast medium faster than the albumin of unmixed serum, suggesting that the protein was bound to the contrast medium. The majority of the protein moved towards cathode. In starch gel electrophoresis, where the gel acts as a microfilter, all serum and contrast medium mixture components migrated slowly, suggesting an increase in the size of the moving particle, indicating aggregation. These changes of electrophoretic migration pattern could be reversed by dialysis, suggesting that the major proteins were not denatured or permanently altered.

Red cell clumping. Clumps of red cells formed when blood was mixed with contrast medium. These clumps were firm and their formation was enhanced by agitation of the mixture. As can be seen in Fig 3 a variety of shapes of

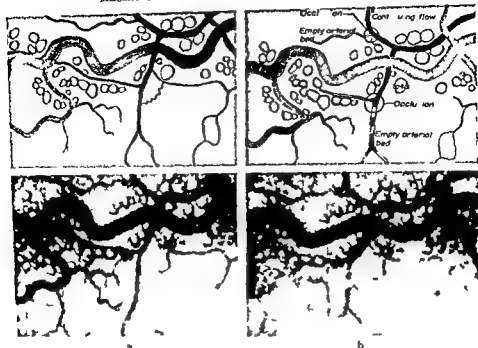


Fig. 4. Frames from a motion film with schematic drawings made of rat's mesentery before (a) and after (b) injection of 6 parts of blood and 4 parts 50% sodium diatrizoate into aortic arch. O—clots and empty arterial beds.

clumps were observed. Some were round or oval, others were elongated strand-like structures and still others were round with elongated narrow processes. In most of these clumps a clear amorphous central core could be demonstrated. This stained blue when bromphenol blue was added to the mixture and could be clearly seen after red cell clumps had been crushed. This indicated that the central core was a flake or strand of protein. The central protein material also stained bluish with Mallory hematoxylin phosphotungstic acid stain (Mallory 1942), suggesting that it contained fibrin. The optimal concentration of contrast medium in blood for the formation of these clumps was 33% by volume.

Clumps were found in every blood sample of patients and controls studied by our technique. However, the number of clumps observed in 25 young healthy individuals ranged from 3 to 7 per chamber with an average of 5 for the whole group. On the other hand, the clump count of a patient on three different days who developed hemiparesis following cerebral angiography was 25, 23 and 28.

We have investigated the possibility that these clumps are capable of obstructing small blood vessels. Rats were used in these studies because their

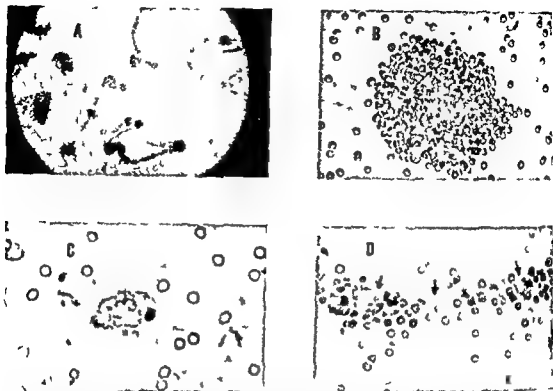


Fig 3 Photomicrographs of clumps formed in a mixture of 7 parts whole blood and 3 parts of 50 sodium dithionite. A) Round, oval and strand like clumps. Magnification 8×25 . B) Round clump showing small clear area left from center. Magnification 8×40 . C) The clear center stained with bromphenol blue. Most of the red cells have been removed by crushing. Increased contrast over the unstained clear area in B. D) Clear strand stained with bromphenol blue joining 2 round clumps with clear centers marked with arrows. Red cells partly removed by crushing.

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Table 2

Comparison of the biological effects of various contrast media

	Clump count in a mixture of 6 parts whole blood and 4 parts of 0.6 molar contrast medium	Number of ml of 0.6 molar contrast medium needed to produce tetany in rats	Coagulation defects in a mixture of 2 parts of plasma and 1 part of 0.6 molar contrast medium	Quick test	Factor 1	Conductivity
Sodium diatrizoate	11	1.5-2.5 ml	8	40		180
Methylglucamine diatrizoate	0	9-11 ml	10	44		42
Sodium iothalamate	9	1.7-2.5 ml	9	40		170
Methylglucamine iothalamate	0	8-10 ml	3	43		46
Sodium acetrizoate	19	0.5-1.5 ml	0	4		200
Iodopaste	17	2.5-3.0 ml	11	22		120

volume clotted slowly and mixtures of 50% by volume did not clot. Addition of calcium ions shortened the Lee-White clotting time somewhat but did not bring it back to normal. Therefore other clotting factors were investigated.

Table 1 shows that several clotting factors and mechanisms were disturbed in a mixture of 7 parts blood and 3 parts sodium diatrizoate. The thrombin clotting time was prolonged 3 or 4 times more than the control. The accelerator globulin (Factor V) activity as estimated by the technique of OWREN was reduced to less than one fifth of that of the control. The activity of barium sulfate adsorbed plasma in the thromboplastin generation test was depressed to less than 1%. This test in part reflects the activities of factors V and VIII.

Comparison of various contrast media. It was noted that the sodium compounds of contrast media constantly gave higher clump counts than the corresponding methylglucamine compounds. Therefore equimolar solutions of available contrast media were prepared and their effects on clump counts, the amount needed to produce tetany in rats, and the disturbance of clotting factors were compared. Table 2 shows that the methylglucamine compounds of diatrizoate and iothalamate produced the least clumps, could be given in the largest amounts before causing tetany, and caused the least disturbances in clotting. The corresponding sodium compounds produced more marked changes.

Table 1

Effects of sodium diatrizoate upon coagulation mechanisms

	Control mixture of 3 parts saline and 7 parts plasma	Mixtures of 3 parts 50 sodium diatrizoate and 7 parts plasma
Thrombin clotting time (TGT)	28 sec	72—90 sec
Accelerator globulin (factor V as assayed by the method of Owren)	68 %	5—10 %
Activity of barium sulfate adsorbed plasma in thromboplastine generation test (TGT factors V, VIII)	Normal	Less than 1 %

blood also formed clumps with the contrast media, and their mesenteric blood flow was easily observed. Fig. 4 shows frames from a motion film made during injection into the aortic arch of a mixture of 6 parts of rat blood and 4 parts of 50 % sodium diatrizoate. Frame (a) was taken before the injection showing the relationship of venous and arterial branchings in the mesentery. Frame (b) was taken of the same field after the injection. There are obstructions visible at 2 bifurcations and empty arterial beds distal to obstructions. While viewing the motion film, one could see stasis in an obstructed arterial branch and continuation of flow in other branches and in the veins. These obstructions could still be seen after 2, 5 and 10 minutes following the injection.

Tetany in rats. During the studies of mesenteric circulation it was noted that the anesthetized rats became hyperexcitable after receiving 1 to 2 ml total of 50 % of sodium diatrizoate or sodium acetrizoate. Touching the rat's nose or whiskers elicited jerking of all extremities and arching of the back. After 4 to 5 ml of contrast medium had been introduced the jerking became spontaneous. This tetanic state could be prevented by mixing 0.2 ml of 10 % calcium chloride solution with 1 ml of contrast medium. Up to 12 ml of such mixtures could be given without the development of tetany. Determinations of combined concentrations of ionized calcium and magnesium in the blood of tetanic rats by EDTA titration demonstrated a depression of these ions by 30 % as compared to control animals. Determination of the combined concentrations of ionized calcium and magnesium in man from blood samples removed serially from the jugular bulb during carotid angiography showed a 25 % reduction during the peak contrast medium concentrations.

Clotting defects. It had been noted that the contrast medium and blood mixtures in which the concentration of contrast medium exceeded 30 % by

Table 2

Comparison of the biological effects of various contrast medias

	Clump count in a mixture of 6 parts whole blood and 4 parts of 0.6 molar medium	Number of ml of 0.6 molar contrast medium needed to produce tetany in rats	Coagulation defects in a mixture of 2 parts of plasma and 1 part of 0.11 molar contrast medium	Q test	Factor I	Conductivity
Sodium diatrizoate	11	1.5-2.5 ml	8 °	40	180	
Methylglucamine diatrizoate	0	9-11 ml	10	44	42	
Sodium iothalamate	9	1.7-2.5 ml	9	40 °	110	
Methylglucamine iothalamate	0	8-10 ml	9	45	46	
Sodium acetrizate	19	0.5-1.5 ml	0	4	200	
Sodium pyracetate	17	2.5-3.0 ml	11	22	120	

volume clotted slowly and mixtures of 50 % by volume did not clot. Addition of calcium ions shortened the Lee-White clotting time somewhat but did not bring it back to normal. Therefore other clotting factors were investigated.

Table 1 shows that several clotting factors and mechanisms were disturbed in a mixture of 7 parts blood and 3 parts sodium diatrizoate. The thrombin clotting time was prolonged 3 or 4 times more than the control. The accelerator globulin (Factor V) activity as estimated by the technique of OWREN was reduced to less than one fifth of that of the control. The activity of barium sulfate adsorbed plasma in the thromboplastin generation test was depressed to less than 1 %. This test in part reflects the activities of factors V and VIII.

Comparison of various contrast media. It was noted that the sodium compounds of contrast media constantly gave higher clump counts than the corresponding methylglucamine compounds. Therefore equimolar solutions of available contrast media were prepared and their effects on clump counts, the amount needed to produce tetany in rats, and the disturbance of clotting factors were compared. Table 2 shows that the methylglucamine compounds of diatrizoate and iothalamate produced the least clumps, could be given in the largest amounts before causing tetany, and caused the least disturbances in clotting. The corresponding sodium compounds produced more marked changes.

Table 1

Effects of sodium diatrizoate upon coagulation mechanisms

	Control mixture of 3 parts saline and 7 parts plasma	Mixtures of 3 parts 50 sodium diatrizoate and 7 parts plasma
Thrombin clotting time (TCT)	28 sec	72-90 sec
Accelerator globulin (factor V) as assayed by the method of Owren)	69 "	5-10
Activity of barium sulfate adsorbed plasma in thromboplastin generation test (TCT factors V, VIII)	Normal	Less than 1 "

blood also formed clumps with the contrast media, and their mesenteric blood flow was easily observed. Fig. 4 shows frames from a motion film made during injection into the aortic arch of a mixture of 6 parts of rat blood and 4 parts of 50% sodium diatrizoate. Frame (a) was taken before the injection showing the relationship of venous and arterial branchings in the mesentery. Frame (b) was taken of the same field after the injection. There are obstructions visible at 2 bifurcations and empty arterial beds distal to obstructions. While viewing the motion film, one could see stasis in an obstructed arterial branch and continuation of flow in other branches and in the veins. These obstructions could still be seen after 2, 5 and 10 minutes following the injection.

Tetany in rats. During the studies of mesenteric circulation it was noted that the anesthetized rats became hyperexcitable after receiving 1 to 2 ml total of 50% of sodium diatrizoate or sodium acetrizoate. Touching the rat's nose or whiskers elicited jerking of all extremities and arching of the back. After 4 to 5 ml of contrast medium had been introduced the jerking became spontaneous. Thus tetanic state could be prevented by mixing 0.2 ml of 10% calcium chloride solution with 1 ml of contrast medium. Up to 12 ml of such mixtures could be given without the development of tetany. Determinations of combined concentrations of ionized calcium and magnesium in the blood of tetanic rats by EDTA titration demonstrated a depression of these ions by 30% as compared to control animals. Determination of the combined concentrations of ionized calcium and magnesium in man from blood samples removed serially from the jugular bulb during carotid angiography showed a 25% reduction during the peak contrast medium concentrations.

Clotting defects. It had been noted that the contrast medium and blood mixtures in which the concentration of contrast medium exceeded 30% by

Table 2

Comparison of the biological effects of various contrast media

	Clump count in a mixture of 6 parts whole blood and 4 parts of 0.6 molar contrast medium	Number of ml of 0.6 molar contrast medium needed to produce tetany in rats	Coagulation defects in a mixture of 2 parts of plasma and 1 part of 0.6 molar contrast medium	Quick test	Factor V	Conductivity
Sodium diatrizoate	11	1.5—2.5 ml	8	40		180
Methylglucamine diatrizoate	0	9—11 ml	10*	44		42
Sodium iothalamate	9	1.7—2.5 ml	9	40*		170
Methylglucamine iothalamate	0	8—10 ml	9*	45		46
Sodium acetrizate	19	0.5—1.5 ml	0	4		200
Iodopyrant	17	2.5—3.0 ml	11	72		120

volume clotted slowly and mixtures of 50 % by volume did not clot. Addition of calcium ions shortened the Lee-White clotting time somewhat but did not bring it back to normal. Therefore other clotting factors were investigated.

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Table 3

Influence of dextran (molecular weight 60 000) upon the biological effect of 0.6 molar sodium diatrizoate

	Sodium diatrizoate	Sodium diatrizoate + 10% dextran
Quick test	7°	14°
Factor V	36"	48"
Clump count	11	3
Amount needed for tetany		
in rats	1.5-2.0 ml	7.5-8.0 ml
Conductivity	175	114

Sodium acetrizoate caused the greatest changes in these factors of this group of contrast media. Iodopyracet, which is chemically less closely related to the media mentioned above, produced large numbers of clumps and markedly altered blood clotting.

Since the sodium compounds showed greater biological effects than the methylglucamine compounds, it was thought that the degree of ionization in these two groups could be different. Therefore, measurements of conductivity of these equimolar solutions were made. As can be seen in column 4 of Table 2, the conductivity of the sodium compounds was higher than the methylglucamine compounds. It was highest in sodium acetrizoate.

Effect of addition of albumin and dextran The methylglucamine compounds which had low conductivity produced less biological effects, i.e. lower clump counts, less tendency to cause tetany, and somewhat smaller disturbances of the clotting mechanism, than sodium salts. It was thought that depression of ionization might be useful in further reducing the biological changes in blood. Since binding of contrast medium with protein was indicated by our earlier experiments we added purified human albumin to contrast media. Albumin added in amounts to produce a concentration of 10 to 15% in the mixture reduced the conductivity, lowered the clump count, increased the amount that could be given before the appearance of tetany, and decreased the defect in clotting. No obstructions were observed in the rat's mesenteric circulation with these mixtures. However, the mixtures were quite viscous with these concentrations of albumin. Dextran was also tested in the hope that it would bind with contrast medium and reduce its ionization and

biological activity Dextran (Dextran Clinical Grade, Henley Co New York, NY) with a molecular weight of 60 000 was found to exert some beneficial effect if added in amounts to make a final concentration of 5 to 15 % Table 3 shows that the biological activity of both the sodium and methylglucamine compounds of diatrizoate were lowered by the addition of dextran The addition of dextran also caused a marked increase in the viscosity of the mixture No obstructions in the rats mesenteric circulation were observed with these mixtures Due to the high viscosity they could only be injected slowly

Discussion

Most water soluble contrast media have biological effects if given intravascularly in extremely large amounts and cause *in vitro* changes if used in high concentrations It is important to know the peak concentrations and the durations of various concentrations during angiographic procedures in humans When the *in vivo* and *in vitro* changes occur at concentrations known to exist in man one may attempt to connect those changes with certain complications

The mere displacement of blood going to the brain by contrast media may be of importance During angiography up to two thirds of the blood in the carotid artery may for a few seconds consist of contrast medium and this may cause a sufficient reduction of oxygen and other products to produce brain dysfunction Individuals particularly susceptible to this situation could be those whose brain already operates under unfavorable conditions such as vascular insufficiency increased intracranial pressure or a severe generalized metabolic disturbance The role of blood displacement by contrast media during angiography in the production of complications has yet to be investigated In instances with rapid circulation through the brain that effect would be negligible but in patients with cerebrovascular disease it could be significant

The changes of electrophoretic mobilities of protein suggest alteration of electrical charges and an increase in the size of the migrating particles This may be a contributory factor in the production of complications in patients whose blood already contains an abnormal protein such as occurs in patients with multiple myeloma and macro-globulinemia These patients are known to tolerate contrast media poorly and they are reported to have developed oliguria or anuria following urography In these patients at autopsy the kidney tubules were found filled with protein casts (HILLMAN et coll 1957 PERRILLIE & COAN 1958) It is currently unknown whether these changes in serum protein are related to the complications of cerebral angiography

The mechanism of depression of calcium and magnesium ions is unclear but may be caused in part by substances added to the contrast medium for anticoagulation stabilization and buffering It also could be caused in part by in

creasing the binding of these ions to the altered protein Tetany in rats was caused by amounts of contrast media which are larger than those used in angiographic procedures in patients. Yet since, *in man*, during arteriography up to two thirds of the intravascular volume can consist of contrast medium, seizures during or immediately following an injection could be related to displacement of blood and binding of these ions.

The formation of red cell clumps *in vitro* has taken place with the blood of every individual tested according to the technique described. We have observed the conjunctival vessels under a microscope of patients during angiography but have not seen definite clumps or permanent occlusions. In rats, mesenteric vessels could be occluded by clumps. It is likely that in most patients during angiography the clumps that may form cause no disturbance, however, in individuals with a marked tendency to form clumps, the microemboli may contribute to the occurrence of complications by blocking the micro circulation of the brain. Such effects would be most marked in a diseased circulatory bed with a slow circulation time.

The coagulation defect is probably caused by binding of contrast medium to coagulation factors. The exact site of action is still unclear. The various clotting factors seem to be affected in different degrees. It is possible that a disturbance is created by interference with polymerization of fibrin monomers. As a result of this, the formation of the normal fibrin network is delayed or does not take place, and abnormal fibrin fragments may develop. These abnormal fibrin fragments may be part of the clear centers of clumps which can be stained with protein stains such as bromphenol blue and by fibrin stains such as Mallory's. The role of a coagulation defect in the production of complications may be twofold. On the one hand it may increase damage by hemorrhage in infarcted areas which may have developed through occlusion by red cell clumps. On the other it may prevent intravascular clotting in areas of stasis and development of propagating thrombi in the collateral channels.

There was a good correlation between the biological effects observed and electrical conductivity of contrast media. Sodium compounds have the higher conductivity, and produced more changes than the methylglucamine compounds, indicating that the latter ionized to a lesser degree. A further reduction of conductivity by the addition of albumin or dextran further reduced the biological effect. However, the addition of albumin or dextran in effective amounts increased the viscosity considerably, making rapid injection difficult. A smaller molecule would be preferable. Since the biological effects seem to be proportional to the degree of ionization, it is likely that they are caused by binding of the ionized contrast medium molecules with protein molecules, perhaps through the carboxyl group of the benzoic acid radical.

It still must be established through further observations whether the biological alterations described by us are actually responsible for complications in patients during angiography. It is of interest that sodium acetrizate and iodopyracet produced the highest clump counts, the greatest disturbance of coagulation and produced tetany with smaller amounts than the diatrizate and iothalamate compounds. Iodopyracet and sodium acetrizate have been abandoned for angiography because of the high incidence of unfavorable reactions.

Acknowledgement

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SUMMARY

Contrast media exert biological effects upon blood components at concentrations that exist in blood vessels during angiography. Effects observed were 1) replacement of blood 2) changes of proteins 3) clumping of red cells 4) depression of ionized calcium and magnesium levels and 5) clotting defects. The possible relationship to complications is discussed. Contrast media studied were sodium diatrizate, methylglucamine diatrizate, sodium iothalamate, methylglucamine iothalamate, sodium acetrizate and iodopyracet. All sodium compounds exerted stronger effects than methylglucamine compounds. Addition of albumin or dextran before use diminished the intensity of biological effects.

ZUSAMMENFASSUNG

Kontrastmittel erzeugen biologische Wirkungen gegenüber Bestandteilen des Blutes bereits in Konzentrationen, die während einer Angiographie in den Blutgefäßen vorhanden sind. Folgende Wirkungen wurden beobachtet: 1) Verdrängung des Blutes 2) Eiweißveränderungen 3) Agglutinierung von roten Blutkörperchen 4) Verringerung des Spiegels von ionisiertem Calcium und Magnesium und 5) fehlerhafte Koagulation. Die mögliche Verwandtschaft dieser Veränderungen mit Komplikationen wird besprochen. Die untersuchten Kontrastmittel sind Natriumdiatrizoat, Methylglukamindiatrizoat, Natriumiothalamat, Methylglukaminiothalamat, Natriumacetrizot und — Iodopyracet. Alle Natriumverbindungen riefen stärkere Wirkungen hervor als die Methylglukaminverbindungen. Zusatz von Albumin oder Dextran vor Gebrauch verminderte die Stärke der biologischen Wirkungen.

RÉSUMÉ

Les moyens de contraste ont des effets biologiques sur les composants du sang à des concentrations qui existent dans les vaisseaux sanguins au cours de l'angiographie. Ces effets sont: 1) le remplacement du sang par le moyen de contraste 2) des modifications des protéines 3) l'agglomération des globules rouges 4) l'abaissement du taux du calcium et du magnésium.

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- S H E I B E R G P and Z U N K E R E Complications in direct percutaneous carotid arteriography
Arch Neurol 11 (1963) 676
- S I T H I E S O Zone electrophoresis in starch gels group variations in the serum proteins of
normal adults Biochem J (1955) 629
- S O B I N S H F R A S I E R W G J A C O B S E N G and V A N E E C K H O V E N F A Nature of adverse
reactions to radiopaque agents preliminary report J Amer med Ass 170 (1959) 1546
- T E R A S I M I D E S T G M A R G O L I S G and P O V T O N H J Prophylaxis of experimental contrast
medium injury to the spinal cord by vasodepressor drugs Angiology 14 (1963) 394
- Z W E I F A C H H W and M E T Z D B Rat mesoappendix procedure for bioassay of humoral
substances acting on peripheral blood vessels Ergebn Anat 35 (1956) 176

ionisés et 5) des lacunes par caillots. Les auteurs examinent les rapports possibles entre ces effets et les complications. Les moyens de contraste étudiés sont : le diatrizoate de sodium, le diatrizoate de méthylglucamine, l'iothalamate de sodium, l'iothalamate de méthylglucamine, l'acétriatoate de sodium et l'iodopyracet. Tous les composés sodés ont des effets plus intenses que les composés de la méthylglucamine. L'addition d'albumine ou de dextran diminue l'intensité des effets biologiques.

REFERENCES

- BAGIMANN I. et coll. Differentiation of the factor V II complex. Studies on the Stuart Prower factor. *Thrombos. Diathes. haemorrh.* 1 (1957) 169.
- BASSETT R. C., ROCHFERS J. S., CHERRY R. C. and GRUSHITT C. The effect of contrast media on the blood brain barrier. *J. Neurosurg.* 10 (1953) 38.
- BROWAN I. and OLSSON O. Experimental study of contrast media for cerebral angiography with references to possible injurious effects of cerebral blood vessels. *Acta radiol.* 31 (1949) 321.
- CULP D. A., VAN EPTS F. F. and EDWARDS C. N. Comparative studies of urographic media. *J. Urol.* 78 (1957) 493.
- EPSTEIN J. A., HOFFMAN I. and EPSTEIN B. S. Electrocardiographic alterations observed during percutaneous cerebral angiography. *Arch. Neurol. Psychiat.* 81 (1959) 142.
- FIELD J., ROBERTSON J. T. and DESVUSSURE R. L. Jr. Complications of cerebral angiography in 2 000 consecutive cases. *J. Neurosurg.* 19 (1962) 775.
- IOLTZ L. F., THOMAS L. B. and WARD A. A. Jr. Effects of intracarotid diodrast. *J. Neurosurg.* 9 (1952) 68.
- GRADWOL R. B. H. Clinical laboratory methods and diagnosis, vol. I. 5th edition p. 440. C. V. Mosby Co. St. Louis 1956.
- GREITZ T. and WEISS S. EFG and EKG in cerebral angiography with sodium diatrizoate (Hypaque) and methylglucamine diatrizoate (mucokon). *Acta radiol.* 52 (1959) 145.
- JOHNSON J. H. and KNISLEY M. H. Intravascular agglutination of the flowing blood following the injection of radiopaque contrast media. *Neurology* 12 (1962) 560.
- KILLMAN S., GJORUP S. and THILSEN J. H. Fatal acute renal failure following intravenous pyelography in patients with multiple myelomata. *Acta med. scand.* 1958 (1957) 43.
- KUTT H. and McDOWELL F. Effects of iodinated contrast media upon electrophoretic mobilities of blood proteins and lipoproteins. *J. Lab. clin. Med.* 59 (1962) 118.
- MILLIOTAT T. H. and McDOWELL F. The effect of iodinated contrast media upon blood proteins, electrolytes and red cells. *Neurology* 13 (1963) 492.
- SCHICK R. W., KENNIE L. E. and McDOWELL F. The quantities of sodium diatrizoate in cerebral vessels during arteriography. *J. Neurosurg.* 20 (1963) 515.
- MALLORY F. B. Pathological technique. 76 W. B. Saunders Co. Philadelphia 1942.
- OWREN P. A. The coagulation of blood: investigations of a new clotting factor. *Acta med. scand. Suppl.* 194 (1947).
- PENDERGRASS E. I. et coll. Further considerations of deaths and unfavorable sequelae following administration of contrast media in urography in the United States. *Amer. J. Roentgenol.* 74 (1955) 262.
- PERILLIE P. II and CONN H. O. Acute renal failure after intravenous pyelography in plasma cell myeloma. *J. Amer. med. Ass.* 167 (1958) 2.
- QUICK A. J. Coagulation defect in sweet clover disease and in the hemorrhagic chick disease of dietary origin. *Amer. J. Physiol.* 118 (1937) 260.



Fig 1 Left anterior posterior angiograms. Lateral displacement of internal carotid artery by aneurysm partially filled with contrast material.

ry contusion with mild pulmonary hemorrhage. The patient's respiratory distress improved but he developed considerable difficulty in swallowing and his voice became weak and hoarse. Laryngoscopy revealed an area of ecchymosis on the left posterior pharyngeal wall, weakness of the left side of the palate and a left vocal cord paralysis. During the postoperative period the left pupil remained constricted. The dysphagia gradually improved and the patient was discharged after 20 days of hospitalization.

Throughout the next five months the left palatal weakness and the left vocal cord paralysis persisted. Three months after discharge the patient began to complain of left occipital headaches and frequent vomiting. At this time a tender mass was palpable high in the left side of the neck and fullness of the left tonsillar and pharyngeal areas was noted. Six milliliters of thick, old blood from the mass was aspirated through the left pharyngeal wall. Shortly thereafter atrophy and fasciculations involving the left side of the tongue were observed. It was felt that the mass probably represented an old hematoma related to the previous trauma.

Physical examination. On admission to The Johns Hopkins Hospital a firm, tender 4 x 4 cm mass high in the left cervical area and partially under the angle of the mandible was palpated by intra and extraoral digital examination. The mass pulsed but it could not be determined whether these pulsations were direct or transmitted. The carotid artery was displaced laterally and anteriorly by the mass. A bruit was heard over the mass. There was some limitation of range of neck motion because of pain, especially with involvement to the left. The neurological examination revealed a left partial Horner's syndrome with miosis and ptosis. The tongue deviated to the left and there was obvious atrophy and fasciculation of the

DISSECTING ANEURYSM OF INTERNAL CAROTID ARTERY AFTER NON-PENETRATING NECK INJURY

Case report

by

MAXWELL D LAI, HARRY B HOFFMAN and JOSEPH J ADAMKIEWICZ

Dissecting aneurysms of the carotid vessels in the neck following penetrating injuries are well known. Non penetrating injuries resulting in vascular damage may lead to thrombosis. The purpose of this paper is to report an unusual case of cervical carotid artery dissecting aneurysm following a hyperextension neck injury sustained in an automobile accident.

Case report

History. A 21 year old white male was admitted to The Johns Hopkins Hospital with a chief complaint of pain in the left side of the head and neck of 8 months duration.

Six months previously he had been admitted to The Baltimore City Hospital some minutes after the automobile he was driving collided with a truck. He was conscious but restless and in a state of shock having a systolic blood pressure of 50 mm Hg. Multiple facial lacerations were noted but there was no evidence of trauma to the neck. Within 30 minutes his left pupil became constricted. Because he was in shock a four quadrant paracentesis was performed and blood was revealed in the left upper quadrant. At exploratory laparotomy a ruptured spleen was found and a splenectomy was performed. A chest film taken in the early postoperative period showed a fracture of the left ninth rib and a left pneumothorax. Because of increased respiratory distress a tracheotomy was performed. Subsequent chest films showed left pulmonary

Discussion

This patient sustained an acute hyperextension neck injury as indicated by the history of an automobile accident, the cervical spine fracture, and lack of evidence of a penetrating injury. The subsequent development of a pulsating mass in the neck with multiple peripheral nerve deficits and a partial Horner's syndrome gave clues to the possibility of formation of a carotid artery aneurysm. The tracheal shift to the left and the relative enlargement of the left pyriform sinus were radiological signs compatible with the clinically known cranial nerve deficits. Carotid arteriography demonstrated the dissecting aneurysm of the internal carotid extending from the common carotid bifurcation to the base of the skull. The location and size of the aneurysm accounted for the involvement of the multiple cranial and cervical sympathetic nerves. Vertebral angiography was necessary to exclude involvement of this artery in the aneurysm and independent vertebral artery thrombosis. The left vertebral artery was small and probably hypoplastic. The superior half of this vessel was not demonstrated and thrombosis could not be excluded. At surgery there was no evidence of vertebral contribution to the aneurysm.

Carotid and vertebral arterial injuries following both penetrating and non-penetrating neck trauma have been reviewed by SEUDIS *et coll* (1962) as well as by GUARDJIAN *et coll* (1963). Dissecting aneurysms of the cervical vessels following penetrating injuries have been well documented. The mechanism of formation consists of a local tear in the vessel wall leading to subintimal or medial dissection and subsequent incomplete or complete occlusion of the vessel lumen. The proximity of the arteries and veins in the neck make possible the formation of arteriovenous fistulas.

Examples of penetrating injuries besides stab or missile wounds of the neck include inadvertent laceration at the time of surgery (DENECKE 1958) and percutaneous carotid angiography (FLEWING & PARK 1959). Such injuries can also lead to thrombotic occlusion of the cervical vessels. Thrombus which forms at the site of intimal tear, may extend by forward propagation to occlude a long segment of the vessel.

Non-penetrating neck injuries more commonly lead to thrombosis than to dissecting aneurysm. As in the case of penetrating injuries, the mechanism consists of a local intimal tear with thrombus formation. This occurred as shown by surgical exploration, in the third case reported by GUARDJIAN in which the patient sustained direct blunt trauma to the left anterior neck region. NORTHGROFT & MORGAN (1944) reported a fatal case of accidental hanging which led to dissection and complete occlusion. Thrombotic occlusion of portions of the vertebral basilar system following chiropractic manipulation has been reported by FORD & CLARK (1956).



Fig 2 Direct injection into aneurysm. Numerous blood clots outlined.

left side of the tongue. The pharyngeal wall sagged on the left and did not rise with phonation although the gag reflex was present. The left vocal cord was completely paralyzed. There was weakness and partial atrophy of the left sternocleidomastoid and trapezius muscles.

Roentgen examination. In the cervical spine a small, non-displaced chip fracture involving the left uncinate process at the posterolateral margin of the upper surface of the C7 vertebra was found. The trachea was shifted to the left and the left piriform sinus was enlarged.

A percutaneous left carotid cerebral angiography revealed considerable lateral displacement of the internal carotid artery in the cervical region by a medially located mass. The mass promptly filled with contrast medium and the lumen of the internal carotid was narrowed as it stretched over the lateral aspect of the mass (Fig 1).

A left selective vertebral angiography performed by a brachial artery catheterization revealed a very small left vertebral artery. Following aspiration of clotted blood from the mass, contrast material was injected directly into the mass to define its limits (Fig 2).

The radiological diagnosis was a dissecting aneurysm of the extracranial portion of the left internal carotid artery extending from the common carotid bifurcation to the base of the skull.

Operation. A 4 x 6 cm dissecting aneurysm of the internal carotid artery was found. The hypoglossal nerve was stretched over the aneurysmal sac. The vagus nerve was completely adherent to its undersurface and the spinal accessory nerve was displaced backward and inferiorly. The superior portion of the aneurysmal sac approached the base of the skull and thus precluded arterial replacement. Therefore the external and internal carotid arteries were ligated and the aneurysmal sac was incompletely removed.

Course. The patient made an uneventful postoperative recovery. He has been followed one year without abnormal neurological findings other than those cranial nerve deficits present prior to surgery.

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REFERENCES

- ANDERSON H McD and SCHLICHTER M M A case of spontaneously dissecting aneurysm of the internal carotid artery J Neurol Neurosurg Psychiat 22 (1959) 195
- DENECKE H J Zur therapie des traumatischen extrakraniellen Aneurysmas der Carotis int im Sauglingsalter mit Recurrens und Vaguslähmung HNO 7 (1958) 119
- FLEMING J F R and PARK A M Dissecting aneurysms of the carotid artery following arteriography Neurology 9 (1959) 1
- FORD F H and CLARK D Thrombosis of the basilar artery with softenings in the cerebellum and brain stem due to manipulation of the neck A report of two cases with one post mortem examination Reasons are given to prove that damage to the vertebral arteries is responsible Johns Hopk Hosp Bull 98 (1956) 37
- GLADJIAN E L HARDY W G LUDNER H W and THOMAS L M Closed cervical cranial trauma associated with involvement of carotid and vertebral arteries J Neurosurg 20 (1963) 418
- NORTHCROFT G B and MORGAN A D A fatal case of traumatic thrombosis of the internal carotid artery Brit J Surg 32 (1944) 105
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An arteriosclerotic plaque is often mentioned as the site of origin of subintimal tear in both penetrating and non penetrating neck injuries. Aortic arch dissections may also propagate into the cervical portion of the carotid arteries. Spontaneous dissecting aneurysm involving the internal carotid artery in a case described by ANDERSON & SCHLICHTER (1959) was explained by the presence of cystic medial necrosis.

There were no findings in this case to suggest an associated vascular abnormality that predisposed the carotid artery to traumatic damage, since a complete review showed that this young patient was in excellent health. The mechanism of formation of the dissection apparently was caused by a marked stretching of the vessel with subsequent intimal tear. Instead of thrombosis, a false passage developed in the vessel wall, leading to the formation of a large dissecting aneurysm. Still, the true lumen persisted to allow filling of the intracranial portion of the internal carotid.

Acknowledgement

We wish to thank Dr Neal Aronson for his kindness in allowing us to use his patient and his encouragement in writing this case report.

SUMMARY

A dissecting aneurysm involving the cervical portion of the left internal carotid artery occurred after acute hyperextension neck injury sustained in an automobile accident. Following the acute injury a pulsating mass associated with multiple peripheral cranial nerve deficits and a partial Horner's syndrome appeared in the neck. The definitive diagnosis was made by carotid angiography and the aneurysm was successfully treated by carotid ligation.

ZUSAMMENFASSUNG

Ein dissezierendes Aneurysma, das den Halsteil der linken Art. carotis int. umfaßt, trat nach akuter Hyperextensionsverletzung des Nackens als Folge eines Autounfalls auf. Im Anschluß an die akute Verletzung trat eine pulsierende Schwellung auf. Sie war begleitet von multiplen peripheren Hirnnervenausfällen und einem partiellen Horner's Syndrom. Die endgültige Diagnose wurde mittels Carotisangiographie gestellt. Das Aneurysma wurde erfolgreich mit Carotisunterbindung behandelt.

RÉSUMÉ

Un anévrysme disséquant de la partie cervicale de la carotide interne gauche est apparu après une hyperextension cervicale brusque au cours d'un accident d'automobile. Après la période aigue du traumatisme, une masse pulsatile apparut dans le cou, accompagnée de déficits périphériques de plusieurs nerfs crâniens et d'un syndrome partiel de Claude Bernard-Horner. Le diagnostic définitif a été fait par l'angiographie carotidienne et l'anévrysme a été traité avec succès par ligature de la carotide.

REFERENCES

- ANDERSON R McD and SCHIECHTER M M A case of spontaneously dissecting aneurysm of the internal carotid artery *J Neurol Neurosurg Psychiat* 22 (1959) 195
- DEVECKE H J Zur therapie des traumatischen extrakraniellen Aneurysmas der Carotis int im Säuglingsalter mit Recurrens und Vaguslähmung *HNO* 7 (1958) 119
- FLEMING J F R and PARK A M Dissecting aneurysms of the carotid artery following arteriography *Neurology* 9 (1959) 1
- FORD F R and CLARK D Thrombosis of the basilar artery with softenings in the cerebellum and brain stem due to manipulation of the neck A report of two cases with one post mortem examination Reasons are given to prove that damage to the vertebral arteries is responsible *Johns Hopk Hosp Bull* 98 (1956) 37
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- NORTHCROFT G B and MORGAN A D A fatal case of traumatic thrombosis of the internal carotid artery *Brit J Surg* 32 (1944) 105
- SPUDIS E V SCHIARYJ M ALEXANDER E and MARTIN J F Dissecting aneurysms in the neck and head *Neurology* 12 (1962) 867

COMPLICATIONS OF DIRECT AND INDIRECT ANGIOGRAPHY OF THE BRACHIOCEPHALIC VESSELS

by

ERICH K. LANG

The advent of surgical therapy for extracranial cerebrovascular disease necessitated new angiographic methods which would permit demonstration of the entire aortic arch and brachiocephalic vessels in various flexion and rotation positions of the head and neck. The advocacy of catheter angiography for optimal demonstration of the structures as well as the increasing popularity of indirect brachial and axillary angiography challenges a comparison of the advantages and safety of these procedures with direct puncture of the carotid artery. This paper intends to evaluate the advantages and disadvantages of these various procedures and compare the relative safety of some 1 800 angiographies after direct puncture of the carotid artery to some 820 indirect angiographies using the brachial or axillary approach and some 11 402 retrograde percutaneous catheter angiographies (9).

Carotid angiography after direct puncture is a technically simple procedure allowing excellent demonstration of the intracranial vessels. While this method offers the best detailed demonstration of these vessels, its chief disadvantage is inability to demonstrate the proximal portions of the brachiocephalic vessels and the aortic arch. Limitation of motion of the head and neck area after needle puncture prevents examination of the carotid and vertebral arteries in certain flexion and rotation positions of the head and neck.

Angiography after puncture of the brachial or axillary artery is likewise a technically simple procedure. Right brachial angiography usually provides complete demonstration of the right carotid, cerebral and vertebrobasilar systems. Left brachial angiography is used for the left vertebrobasilar system. Demonstration of the left common carotid artery, however, is quite difficult with this method and is accomplished in less than one third of all attempts. The main disadvantage of this technique is the necessarily large amount of contrast medium which has to be injected to allow for proper filling of the aortic arch and the brachiocephalic vessels. Isotope flow studies suggest a flow to the vertebral artery exceeding 25 % of the total amount injected whenever in excess of 30 ml of volume (10). Because of intolerance of brain to high concentrations of iodinated contrast media, substances used in this area have to be kept to less than twenty eight volume percent iodine content to avoid severe convulsive reactions. These relatively low density contrast media render proper demonstration of the aortic arch difficult (12).

Retrograde percutaneous catheter angiography offers by far the most complete examination of this area (9). All brachiocephalic vessels can be engaged selectively and demonstrated in various flexion and rotation positions of the head and neck. Homogeneous filling of the entire aortic arch with contrast medium is readily achieved by injection of a highly iodinated contrast medium into the ascending aorta. Completely free motion of the head and neck area during the examination allows reproduction of any position that may aggravate or provoke the patient's condition. The disadvantage of this method is the somewhat more complicated technique and time consuming procedure.

Regardless of the risks involved, certain conditions will dictate the preferential use of one or the other method. Direct carotid needle angiography renders the best detail demonstration of the intracranial vessels. Patients suspected of having vascular insufficiency, and particularly if symptoms become manifest in certain positions are best examined by retrograde catheter angiography. This method alone is capable of answering all pertinent questions and producing a satisfactory demonstration of the arterial system in various extreme positions. Indirect angiography from the brachial or axillary artery is particularly useful for the demonstration of the vertebrobasilar system. The amount of contrast medium reaching the vertebrobasilar system can be carefully gauged and overflooding avoided. The method is also used for panarteriography of the aortic arch.

The choice of contrast medium is governed by the injection site. Direct carotid or vertebral needle angiography is best carried out with 50 % diatrizoate sodium solution (Hypaque 50). 60 % meglumine iothalamate (Conray) or sodium and methylglucamine diatrizoates (Renovist). The amount of

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also the risks of the various procedures deserve to be evaluated properly. The incidence rate of complications has varied greatly in various reported series (Table 4). This undoubtedly, depends on the quality of follow up and upon what each individual author chose to interpret as a complication (1, 3, 4, 5, 6, 13, 14, 15, 16, 17, 18).

An analysis of causative factors of complications readily identifies three factors: pre-existing conditions, faulty technique, and complications associated with tissue toxicity of the contrast medium.

For the purpose of comparative study, complications are grouped into fatal complications, serious complications, and minor complications. Minor complications differ from the more serious complications only quantitatively by their short duration and absence of late sequelae. Serious complications may be severe reactions of transient nature or reactions resulting in a permanent deficit.

Analysis of the material in the present series as well as previously reported series reveals certain common denominators. Fatal reactions appear to be commonly linked to the underlying disease process. The contributing force of the angiography to the ultimate demise of the patient remains questionable in many instances.

Serious complications such as focal or generalized seizures, hemiparesis, hemiplegia, unilateral dysesthesias, visual field defects, and language disorders of transitory or permanent nature appear to be greatly influenced by underlying vascular insufficiency and the amount and concentration of contrast medium used. The potentiating effect of these two factors is also readily seen.

The tissue toxicity of contrast media is related to their capillary concentration and preceding trauma to the effector tissue. Hence vascular insufficiency will not only increase the capillary concentration but also render the effector tissue more susceptible.

Patients with primary or metastatic brain tumors show a specific reaction to contrast media injection. This may well be related to a change in the blood brain barrier in the tumor area and a specific reaction to the contrast medium.

Focal and generalized convulsions appear to be directly related to the amount and concentration of contrast medium in the effector area. Hence the amount of each injection, the multiplicity of injections, and the cerebral flow rate appear to be the main determining factor predisposing to such reactions. Subintimal injections of contrast medium and resulting spasm of the arteries will tend to decrease the cerebral flow and hence increase the concentration of contrast medium in the capillary phase. This is probably the mechanism by which convulsions may result from this technical difficulty. (2) Transient aphasia and hemiparesis are likewise commonly seen following a subintimal

Table 1

*Complications in 11 402 Seldinger procedures (Reprint from Radiology Vol 81 (1963) pp 257-263
Survey of the complications of percutaneous retrograde arteriography)*

Fatal complications	7
Serious complications	31
Arterial thrombosis	47
(with secondary loss of limb)	6
Tip of guide wire or catheter broken	5
Perforation of major vessels with serious complications	13
Renal complications	2
Bowel ileus and necrosis	3
Minor complications	375
* Perforation of a major vessel with sequelae	27
* Intramural or subintimal contrast injection without sequelae	136
* Local hematomas	167

* Temporary arterial spasm and loss of pulse were also observed in practically all patients with minor complications

contrast medium used for the injection of each carotid artery should not exceed 12 to 15 ml and 6 ml of contrast medium should be the upper limits for vertebral studies

Indirect angiography is likewise best performed with media of low iodine content. The amount used for each injection is considerably larger since a significant dead space is filled prior to overflow into the vertebral or carotid system. Amounts up to 50 ml can be safely used. Selective catheter angiography is also best performed with media of low iodine concentration. The maximum amounts are similar to the recommended dosage in direct needle angiography. Media of high iodine concentration such as sodium iothalamate 80 % (Angio Conray) and sodium and methylglucamine diatrizoates 90 % (Hypaque M 90) should only be used for injection into the ascending aorta and studies of the aortic arch. Tissue toxicity and a specific effect upon the blood brain barrier limit the use of media of higher iodine concentration in the head and neck area (7).

Local anesthesia is usually preferred for indirect brachial and axillary angiography and retrograde percutaneous catheter angiography. General anesthesia is commonly used for direct needle carotid and vertebral arteriography.

Complications

The literature on cerebral angiography has repeatedly emphasized the values as well as the dangers of this diagnostic technique. Since a choice of several techniques is offered to the physician not only their advantages but

Table 2 (cont.)

Reactions occurring in different angiographic examinations	Vertebral (430 cases)*	Aortic arch (320 cases)	Descending aorta (60 cases)
<i>Serious complications</i>			
Arterial thrombosis	4	4	—
Loss of limb	1	1	—
Convulsions	11	37	12
Large hematoma	1	2	7
Petechial skin hemorrhage	2	8	7
Ulner parestia	1	—	—
Median nerve paresis	2	—	—
Pseudo-aneurysm at puncture site	2	1	1
Dissecting aneurysm of the brachial or the a. iliac artery	2	1	2
Subcutaneous dye injections	41	14	4
Extravasation of dye into the perivascular space	4	1	—
Pain persisting over 24 hours	3	4	2
<i>Minor complications</i>			
Hematoma	38	43	11
Temporary loss of pulse	382	286	60
Causalgia	1	—	2

2 deaths — Aneurysm of the basilar artery and cerebellar medulloblastoma

* Associated with perivascular extravasation of dye

patients with brain tumors. This is felt to represent a well known reaction of patients with brain tumors to contrast media injection. Focal or generalized convulsions were seen in 7 patients where inadvertent injection of the vertebrobasilar system with 12 ml of contrast medium was carried out. In 32 other patients a review of the films revealed either pre-existing extensive arteriosclerotic disease with vascular impairment or subintimal injection resulting in vascular spasm. Hence a decreased cerebral flow rate and increased iodine content in the capillary phase is incriminated for the convulsive reaction. Temporary hypoxia likewise may have contributed to this reaction. In the third group — complications secondary to faulty technique — arterial thrombosis, abscess formation, formation of large retropharyngeal hematomas and a thyrotoxic storm could be directly related to multiple traumatic punctures or faulty placement of the needle.

Minor complications were numerous and most commonly caused by faulty

Table 2

Retrograde needle angiographies (820) (Survey compiled from 31 completed returns) In all cases the brachial artery or axillary artery was punctured with a No 17 or 18 thin walled needle

Type of examination	Patients	Contrast media	Amount ml	Pressure injection
A Vertebral angiography	430	Diatrizoate sodium 50 % (Hypaque 50) sodium iothalamate 80 % (Angio Conray) meglumine iothalamate 60 % (Conray) sodium and methylglucamine diatrizoates (Renovist)	15 to 30	Occasionally
B Aortic arch angiography	325	Diatrizoate sodium 50 % (Hypaque 50) sodium & methylglucamine diatrizoates (Hypaque M 90 %) sodium iothalamate 80 % (Angio Conray) meglumine iothalamate 60 % (Conray) sodium & methylglucamine diatrizoates (Renovist) diatrizoate methylglucamine 85 % (Cardiografin)	30 to 60	Yes
C Iliac arteriography	65	Diatrizoate sodium 50 % (Hypaque 50) sodium and methylglucamine diatrizoates (Hypaque M 90 %) sodium and methylglucamine diatrizoates (Renovist) sodium iothalamate 80 % (Angio-Conray) diatrizoate methylglucamine (Renografin 60) Diatricon	50 to 80	Yes

injection of contrast medium and are probably direct consequences of the decreased cerebral flow rate and relative anoxemia

Carotid angiography after direct puncture The fatal complication rate in this series is slightly greater than 2 %. However, the majority of these complications are probably not related to the angiographic procedure. The underlying condition was such that the demise of the patient could be expected (Table 3)

The serious complications are readily divided into three groups. Somnolence, progression of neurologic symptoms such as visual field defects, language disorders, hemiplegia and hemiparesis were observed in a large number of

Table 2 (cont.)

Reactions occurring in different angiographic examinations	Vertebral (430 cases)*	Aortic arch (375 cases)	Descending aorta (73 cases)
<i>Serious complications</i>			
Arterial thrombosis	4	4	—
Loss of limb	1	1	—
Convulsions	11	37	12
Large hematoma	1	2	7
Petechial skin hemorrhage	2	8	7
Ulnar paresthesia	1	—	—
Median nerve paresthesia	2	—	—
Pseudo-aneurysm at puncture site	2	1	1
Dissecting aneurysm of the brachial or the subclavian artery	2	1	2
Subintimal dye injections	41	14	4
Extravasation of dye into the perivascular space	4	1	—
Ischemia persisting over 24 hours	3	4	2
<i>Minor complications</i>			
Hematoma	38	43	6
Temporary loss of pulse	38†	28†	60
Causalgia	1	—	—

* 2 deaths — Aneurysm of the basilar artery and cerebellar medulloblastoma

†† Associated with perivascular extravasation of dye

patients with brain tumors. This is felt to represent a well known reaction of patients with brain tumors to contrast media injection. Focal or generalized convulsions were seen in 7 patients where inadvertent injection of the cerebrobasilar system with 12 ml of contrast medium was carried out. In 32 other patients a review of the films revealed either pre-existing extensive arteriosclerotic disease with vascular impairment or subintimal injection resulting in vascular spasm. Hence a decreased cerebral flow rate and increased iodine content in the capillary phase is incriminated for the convulsive reaction. Temporary hypoxia likewise may have contributed to this reaction. In the third group — complications secondary to faulty technique — arterial thrombosis, abscess formation, formation of large retropharyngeal hematomas and a thyrotoxic storm could be directly related to multiple traumatic punctures or faulty placement of the needle.

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Type of examination	Patients	Contrast media	Amount ml	Pressure injection
A Vertebral angiography	130	Diatrizoate sodium 50% (Hypaque 50) sodium iothalamate 80% (Angio Conray) meglumine iothalamate 60% (Conray) sodium and methylglycamine diatrizoates (Renovist)	15 to 30	Occasionally
B Aortic arch angiography	325	Diatrizoate sodium 50% (Hypaque 50) sodium & methylglucamine diatrizoates (Hypaque M 90%) sodium iothalamate 80% (Angio Conray) meglumine iothalamate 60% (Conray) sodium & methylglucamine diatrizoates (Renovist) diatrizoate methylglucamine 85% (Cardiografin)	30 to 60	Yes
C Intrarterio-gram	65	Diatrizoate sodium 50% (Hypaque 50) sodium and methylglucamine diatrizoates (Hypaque M 90%) sodium and methylglucamine diatrizoates (Renovist) sodium iothalamate 80% (Angio Conray) diatrizoate methylglucamine (Renografin 60) Di-Triocin	50 to 80	Yes

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Reactions occurring in different angiographic examinations	Vertebral (430 cases)	Aortic arch (375 cases)	Descending aorta (65 cases)
<i>Serious complications</i>			
Arterial thrombosis	4	4	—
Loss of limb	1	1	—
Convulsions	11	37	17
Large hematoma	1	2	7
Perichestal skin hemorrhage	2	8	7
Unilateral paresis	1	—	—
Median nerve paresis*	2	—	—
Pseudo-aneurysm at puncture site	—	1	1
Dissecting aneurysm of the brachial or the axillary artery	2	1	2
Subintimal dye injections	41	14	4
Extravasation of dye into the perivascular space	4	1	—
Pain persisting over 24 hours	3	4	2
<i>Minor complications</i>			
Hematoma	36	43	11
Temporary loss of pulse	382	286	60
Causalgia	1	—	—
2 deaths — Aneurysm of the basilar artery and cerebellar medulloblastoma associated with perivascular extravasation of dye			

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Minor complications were numerous and most commonly caused by faulty

Table 3

Complications encountered in 1 800 carotid angiographies all performed under general anesthesia (60% were bilateral) Direct puncture technique with a modified Couard No. 17 or 18 needle (long bevelled needles were used from 1951 through 1961 short bevelled since 1961) Hypaque 50% was used 14 ml for unilateral and 28 ml for bilateral injections

Death within 48 hours following procedure

(none appeared to be necessarily related to the angiographic procedure although 5 tumor cases showed rapid progression of neurologic deficit following the examination)

Aneurysm	23
Multiple trauma	8
Primary brain tumor	4
Metastatic brain tumor	3

Serious complications

Arterial thrombosis (at puncture site)	9
Somnolence and progression of neurologic symptoms (92 of these were tumor cases)	96
Convulsions (7 inadvertent vertebral injections)	47
Abscess	1
Large hematoma (tracheotomy necessary)	1
Thyrotoxic storm (accidental injection into the superior thyroidal artery)	1

Minor complications

Total number of extravasations	247
a) Subintimal injections	147
Without sequelae	137
Convulsions	3
Progression of neurologic symptoms (12 tumor cases)	14
Reversal of flow via the anterior communicating artery following a subintimal injection	27
Pain	110
b) Periarterial extravasation	124
Pain (23 with no comment on the presence or absence of pain in the clinical record)	101
Torticollis spasticus	1

placement of the needle. Particularly the long bevelled needle would often transfix the posterior wall of the carotid artery and result in subintimal contrast medium injections. Usually no late sequelae resulted. In many instances, the symptoms of the patient may have been of such minor significance that no specific record was made although some temporary transient neurologic degradation may have been present. Convulsions were recorded in only 3% of the patients and these were minor in nature. A reversal of flow via the anterior communicating artery following subintimal contrast injection into one carotid artery was observed in 22 patients. This is felt to emphasize the hemodynamic significance of subintimal injections resulting in significant reduction of flow in the respective system. Pain was the most common manifestation of

Table 4

Complications from cerebral angiography in the literature

Authors	Angio-gram obtained	Anes-thesia	Mil l	Complica-tions Severe	Fatal	Contrast medium used
FIELD (6)	233?	9?	Local 33	8	8	Hypaque 40
BILL (3)	1000	Not stated	Not stated	Not stated	3	Iodopyracet
SEGELOV (16)	660	{ General 1/3 Local 2/3	7	0	0	Diodrast 30 Urokon 30
CODDEN & KRIEGER (4)	546	Not stated	44	57	8	Diodrast 30 Urokon 70
WHITELEATHER (18)	7300	Local	0	2	0	Hypaque 50
PEREZ (13)	731	{ General 1/4 Local 3/4	27	28	7	Diodrast 30
DUNMORE (5)	147	Local	6	5	3	Diodrast 30
ABBOTT (1)	174	Not stated	2	17	5	Diodrast 30
SEDEWIR (15)	273	General 1	0	0	0	Iodopyracet 47.5
SCHLIMBERG (14)	90?	Local	Not stated	31	3	Hypaque 40

subintimal injections observed in roughly 80% of the patients. Periarterial extravasation was again most commonly associated with faulty technique and use of long bevelled needles. It is felt that the posterior wall of the artery was in most instances perforated during an attempt to advance and thread the needle. Flow was retained since only a portion of the bevelled needle had actually advanced beyond the lumen of the vessel. Pain was the most common and uniform complaint. With the exception of one patient who developed a torticollis spasticus no significant sequelae could be attributed to extravasation of contrast medium.

Indirect angiography The fatal complications appear to be primarily related to underlying pre-existing conditions.

The serious complications are readily divided into two large groups. The first group is related to faulty technique. Arterial thrombosis, loss of limb formation of hematomas ulnar and median nerve paresis pseudo aneurysm at the puncture site dissecting aneurysms of the brachial or axillary artery.

and subintimal injections are the complications encountered in this group. It is noted that, although the use of imaging guide wires and catheters is eliminated by this technique, a substantial rate of arterial thrombosis is still observed. Undoubtedly, this is related to the experience of the operator and pre-existing arteriosclerotic conditions of the vessels.

The most significant complication appeared to be focal or generalized convulsions. The rate of convulsive reactions appeared to be directly proportional to the amount of contrast material used and hence to the concentration in the capillary phase of the vertebrobasilar system. Vertebral angiography utilizing a relatively small amount of contrast medium showed a rate of convulsive reactions of slightly over 2%. Arteriography of the aortic arch, using an intermediate amount of 30 to 60 ml medium of low iodine concentration showed a rate of convulsive reactions of 11.5%. Puncture arteriography, using up to 80 ml medium of high iodine concentration showed 18% convulsive reactions. This appears to establish a causative relationship of relative concentration of iodine to convulsive reactions.

Minor complications were primarily related to local trauma to the arteries and inadequate compression of the puncture site with resulting hematoma formation.

Percutaneous retrograde catheter angiography. The fatal complication rate of 0.06% in this survey is directly related to complications resulting from the angiographic procedure. This series encompasses angiograms of the head and neck area, the aortic arch, as well as the abdominal aorta. Fatal complications seen in a series primarily dealing with abdominal aortography will be less numerous since the common fatalities of brain tumors and cerebral aneurysms will not tend to weight such a series.

The serious complications are, likewise, primarily related to faulty technique and are not weighted by complications associated with primary vascular insufficiency of the cerebrovascular circulation, specific contrast medium complications of these areas, and specific complications seen with brain tumors. However, the common complication of arterial thrombosis at the puncture site using various techniques can be readily compared. Arterial thrombosis was seen in 0.11% with direct needle puncture of the carotid artery and 0.4% with Seldinger needle puncture for introduction of a percutaneous catheter, and in 1% with indirect angiography after needle puncture of the brachial or axillary artery. Specific complications associated with the use of guide wire and catheters are arterial embolism secondary to dislodgement of arteriosclerotic plaques with the guide wire or catheter and breaking of tips of guide wires or catheters within the lumen of the vessel. Arterial

thrombosis is usually related to a combination of underlying vascular disease, poor vascular fitness and multiple traumatic punctures

Minor complications of this procedure are almost exclusively related to faulty technique such as multiple traumatic puncture poor local anesthesia traumatic advancement of the guide wire or catheter, or excessively long catheterization and inadequate compression of the puncture site after completion of the procedure

Major and serious complications can be avoided by careful selection of patients for these procedures and exclusion of patients with low output failure and poor vascular fitness, as well as early institution of anticoagulation therapy in some instances where the patient may be prone to arterial thrombosis at the puncture site because of predisposing conditions

Comments A critical analysis of the data reveals great difficulty in comparing the various figures. The selective use of the various modalities governed by preexisting conditions groups the patient material according to underlying disease entities. However it appears that direct carotid and vertebral needle angiography is an extremely safe procedure in the hands of the experienced operator. The serious complication rate of arterial thrombosis at the puncture site 0.11 % compares well to 0.4 % seen in retrograde catheter angiography and 1 % in indirect angiography. The increased complication rate of direct carotid angiography in patients in the older age group (6 Table 3) is undoubtedly related to arteriosclerotic disease and resulting difficulty in placement and threading of the needle. Retrograde catheter angiography by selective techniques and indirect angiography will circumvent the difficulty of puncturing a severely diseased artery. It is felt that a reduction of the injection flow rate and the amount of contrast material is also necessary to substantially decrease the complication rate in this group since only a combination of atraumatic introduction of contrast medium as well as maintenance of a safe capillary concentration will prevent convulsive reactions

Recommendations Carotid or vertebral angiography after direct puncture is recommended for the demonstration of the intracerebral vessels in detail

Retrograde percutaneous catheter angiography is recommended for selective demonstration of the brachiocephalic vessels in all patients with primarily arterial insufficiency syndromes and particularly those subject to positional aggravation. Indirect angiography after puncture of the brachial or axillary artery may be substituted for this technique if severe arteriosclerotic disease renders the safe passage of catheters via the femoral artery difficult

The injection rate and amount as well as the concentration of contrast

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Minor complications were primarily related to local trauma to the arteries and inadequate compression of the puncture site with resulting hematoma formation.

Percutaneous retrograde catheter angiography. The fatal complication rate of 0.06% in this survey is directly related to complications resulting from the angiographic procedure. This series encompasses angiograms of the head and neck area, the aortic arch, as well as the abdominal aorta. Fatal complications seen in a series primarily dealing with abdominal aortography will be less numerous since the common fatalities of brain tumors and cerebral aneurysms will not tend to weight such a series.

The serious complications are, likewise, primarily related to faulty technique and are not weighted by complications associated with primary vascular insufficiency of the cerebrovascular circulation, specific contrast medium complications of these areas, and specific complications seen with brain tumors. However, the common complication of arterial thrombosis at the puncture site using various techniques can be readily compared. Arterial thrombosis was seen in 0.11% with direct needle puncture of the carotid artery and 0.4% with Seldinger needle puncture for introduction of a percutaneous catheter, and in 1% with indirect angiography after needle puncture of the brachial or axillary artery. Specific complications associated with the use of guide wire and catheters are arterial embolism secondary to dislodgment of arteriosclerotic plaques with the guide wire or catheter and breaking of tips of guide wires or catheters within the lumen of the vessel. Arterial

atteints de tumeurs cérébrales présentent souvent à la suite de l'angiographie une aggravation de leurs signes neurologiques. On pense que les réactions convulsives sont dues à la concentration relative en iode dans le ba hypaque et sont donc influencées par toute réduction du débit due à l'artériosclérose grave ou à toute autre cause. La prévention de l'injection sous l'anima évite des modifications brusques du débit. L'auteur recommande l'angiographie carotidienne ou vertébrale directe pour l'étude des vaisseaux intracérébraux. Il réserve l'angiographie percutanée et trograde par cathétérisme au diagnostic des vaisseaux brachio-céphaliques soumis à des modifications posturales.

REFERENCES

1. ABBOTT K. H. GAY J. R. and FOODALL R. J. Clinical complications of cerebral angiography. *J Neurosurg* 9 (1952) 258.
2. BRENDLER S. J. and HAYES G. J. Hypaque in cerebral angiography. Report of complications in 617 angiograms. *J Neurosurg* 16 (1959) 454.
3. BULL J. W. D. Cerebral angiography. *Postgrad med J* 26 (1950) 157.
4. CODDEN D. R. and KRUEGER H. P. Circumstances surrounding complications of cerebral angiography. Analysis of 546 consecutive cerebral angiograms. *Amer J Med* 25 (1958) 580.
5. DUNNIGRE R. SCOVILLE W. B. and WHITCOMB B. B. Complications of angiography. *J Neurosurg* 8 (1951) 110.
6. FIELD G. ROBERTSON C. T. and DESAUSSEUR G. R. Complications of cerebral angiography in 2 000 consecutive cases. *J Neurosurg* 19 (1962) 775.
7. FLEWING G. P. ROSS A. and PARK A. Dissecting aneurysms of the carotid artery following arteriography. *Neurology* 9 (1959) 1.
8. FUJII K. GRAYSON T. MARGALLIS A. R. and SALTZSTEIN S. Z. The effects of intra arterial injection of contrast media on canine intestine. *Amer J Roentgenol* 89 (1963) 730.
9. LANG E. K. Complications of percutaneous retrograde arteriography. *Survey Radiology* 81 (1963) 257.
10. — External internal carotid artery anastomosis and its correlation to RISA circulation studies. *Radiology* To be published.
11. LATHIER F. R. WEBSTER J. L. and GURDJIAN E. S. Complications of cerebral arteriography. *Case Hosp Bull (Detroit)* 30 (1959) 87.
12. MARSHALL T. R. LING J. T. and COVZALES R. Additional experience with direct percutaneous noncatheter brachial arteriography — left panarteriography — right cerebral arteriography. *Radiology* 81 (1963) 568.
13. PERLLE D. M. LITZ W. C. BEDELL A. J. and CAMPBELL E. Complications following cerebral angiography. *Arch Neurol Psychiat (Chicago)* 71 (1954) 103.
14. SCHLEINBERG P. and ZUCKER E. Complications of direct percutaneous carotid arteriography. *Arch Neurol* 8 (1963) 676.
15. SEDVIN P. C. B. Towards safer angiography. *J Neurosurg* 12 (1955) 460.
16. SEGELOV J. V. Safe angiography. *J Neurosurg* 13 (1956) 567.
17. SILVERSTEIN A. and KRUEGER H. P. Complications of cerebral angiography. A supplementary report. *J Mt Sinai Hosp* 27 (1960) 1.
18. WITTELEATHER J. E. and DESAUSSEUR R. L. Experience with a new contrast medium (Hypaque) for cerebral angiography. *Radiology* 67 (1956) 537.

medium, should be correlated to the injection site, anticipated carotid, cerebral, or vertebro-cerebral flow rates, underlying arteriosclerotic disease, and pre existing conditions such as brain tumors

Overinjection of the vertebral system should be avoided

Meticulous and relatively atraumatic puncture of all vessels should be practiced

Proper threading of short bevelled needle with the bevelled slant pointing to the posterior carotid wall should be used for carotid angiography to prevent subintimal injections, extravasation, and resulting changes in the arterial flow rate of the affected artery

SUMMARY

The advantages disadvantages and complications of direct carotid retrograde percutaneous catheter and retrograde needle angiography are compared and analyzed. The fatal complications seen are usually related to pre existing conditions such as aneurysms brain tumors and so on. Patients with brain tumors often show aggravation of neurologic symptoms following angiography. Convulsive reactions are felt to be related to the relative iodine concentration in the capillary bed and hence are influenced by any reduction of flow rate due to severe arteriosclerotic disease or other causes. Prevention of subintimal injection will safeguard against sudden changes in flow rate. Direct carotid or vertebral angiography is recommended for the demonstration of the intracerebral system. Retrograde percutaneous catheter angiography is reserved for the diagnosis of the brachiocephalic vessels subject to positional changes.

ZUSAMMENFASSUNG

Die Vor- und Nachteile und Komplikationen von perkutanem retrogradem Carotiskatheter und retrograder Nadelangiographie werden verglichen und analysiert. Tödliche Komplikationen sind gewöhnlich zu bereits vorher existierenden Befunden wie Aneurysmen Hirntumoren und Ähnlichem in Beziehung zu setzen. Patienten mit Hirntumoren zeigen oft Aggravation von neurologischen Symptomen im Anschluss an eine Angiographie. Konvulsive Reaktionen scheinen mit der relativen Jodkonzentrationen in den Kapillaren in Beziehung zu stehen und werden durch jede Verminderung der Blutzirkulation infolge ernster Arteriosklerose oder anderen Ursachen beeinflusst. Die direkte Carotis- und Vertebralisangiographie wird zur Darstellung des intracerebralen Gefäßsystems empfohlen. Die retrograde percutane Katheterangiographie ist für die Diagnostik im brachiocephalen Gefäßgebiet das Lageveränderungen unterworfen ist reserviert.

RÉSUMÉ

L'auteur examine et compare les avantages les inconvénients et les complications de l'angiographie carotidienne directe par cathétérisme percutané rétrograde et par ponction rétrograde à l'aiguille. Les complications mortelles sont habituellement rattachées à des affections pré existantes tels que les anévrismes les tumeurs cérébrales etc. Les malades



Fig 1 Angiographie carotidienne droite. Partiellement carotidienne à l'origine d'un angiome étendu à la fosse cérébrale postérieure et à l'hémisphère cérébral droit

Fig 2 Angiographie vertébrale profil. Angiome du vermis

La ventriculographie offrirait pour OLIVEROVA un aspect parfois évocateur image d'allure marécageuse avec un défaut d'injection du 4ème ventricule

Dans les deux cas où nous l'avons pratiquée la ventriculographie gazeuse a montré une dilatation ventriculaire globale avec un défaut d'injection du 4ème ventricule et un refoulement latéral de l'aqueduc de Sylvius. Les 2 fois l'intervention a confirmé qu'un hématome intra-cérébelleux était à l'origine de ces modifications radiologiques

Le bilan angiographique porte à la fois sur les systèmes vertébral et carotidien

L'angiographie carotidienne nous paraît être l'examen à pratiquer d'emblée en raison de sa simplicité et de l'importance des renseignements apportés. Ceux-ci sont de 2 ordres

- 1 Elle objective la participation carotidienne à un angiome étendu non seulement à la fosse cérébrale postérieure mais à tout un hémisphère (Fig 1)
- 2 Dans un nombre non négligeable de cas elle permet de soupçonner l'existence d'une malformation de petit volume bien localisée et incite à pratiquer une angiographie vertébrale. Nous avons rencontré cette éventualité à 4 reprises

Dans le premier cas l'injection de la cérébelleuse supérieure et de la cérébrale postérieure dessinait déjà partiellement l'angiome circumpédonculaire

Dans le deuxième cas la cérébelleuse supérieure légèrement injectée par l'angiographie carotidienne montrait une tache floue dans un hémisphère cérébelleux suffisante pour prévoir la lésion

ASPECTS RADIOLOGIQUES DES ANGIOMES DE LA FOSSE CÉRÉBRALE POSTÉRIEURE

par

J LECUIRE, P BUFFARD, C LAPRAS, A GOUTELLE, J DUQUESNAY, D MICHEL,
G FISCHER et J P DECHAUME

Les angiomes vrais ou anévrismes artérioveineux de la fosse cérébrale postérieure sont moins bien connus que ceux de l'étage sus tentorial, mais le développement des techniques neuro radiologiques et la pratique courante de l'angiographie vertébrale permettent de préciser leurs caractères

A propos d'une série de 11 angiomes de la fosse cérébrale postérieure observés à Lyon au cours de ces 9 dernières années, nous avons tenté d'apprécier la valeur diagnostique des diverses investigations radiologiques : radiographie simple du crâne, ventriculographie, angiographie carotidienne et surtout vertébrale

Les signes fournis par la radiographie simple ont été nettement précisés par HOARE à propos de 3 cas : 1) accentuation des empreintes vasculaires dans la partie postérieure de la voûte osseuse, 2) élargissement des orifices vasculaires (canal transversaire de l'atlas et de l'axis, trou déchiré postérieur)

Dans nos observations, l'étude de ces modifications ne nous a pas fourni de renseignements de valeur



Fig 1 Angiographie carotidienne droite. Participation carotidienne à l'irrigation d'un angiome étendu à la fosse cérébrale postérieure et à l'hémisphère cérébral droit.



Fig 2 Angiographie vertébrale profil. Angiome du vermis.

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Dans le deuxième cas la cérébelleuse supérieure légèrement injectée par l'angiographie carotidienne montrait une tache floue dans un hémisphère cérébelleux, suffisante pour prévoir la lésion.



Fig. 3 Angiographie vertébrale profil Angiome d'un hémisphère cérébelleux



Fig. 4 Angiographie vertébrale profil Angiome étendu à la fosse cérébrale postérieure et à l'hémisphère droit

Dans le troisième cas, la cérébelleuse moyenne visible sur l'arteriographie carotidienne injectait en partie un angiome cérébelleux.

Dans le dernier cas enfin, l'existence d'une anastomose carotidobasilaire expliquait la vascularisation d'un angiome circumpedunculaire sur l'angiographie carotidienne.

3 Enfin l'angiographie carotidienne apporte des données complémentaires très importantes lorsqu'elle permet d'étudier le jeu des anastomoses, les possibilités de suppléance et l'importance des pédicules issus des territoires sus-tentoriels.

L'angiographie vertébrale reste cependant l'examen qui objective avec le plus de précision les angiomes sous-tentoriels.

Elle peut être pratiquée selon diverses techniques que nous avons toutes utilisées : injection directe percutanée au niveau du canal transversaire, injection rétrograde par voie sous-clavière ou par cathétérisme selon la technique de Seldinger.

L'aspect de l'angiome est commun aux autres malformations cérébrales de même type, avec des pédicules artériels dilatés et souvent une dilatation du tronc de la vertébrale, un peloton vasculaire anormal et des veines de drainage dilatées et flexueuses, précocement injectées.

Il nous semble possible d'individualiser deux formes radiologiques qui correspondent aux formes anatomiques.

1 Les angiomes localisés aux hémisphères cérébelleux (Fig. 3) ou au vermis (Fig. 2).

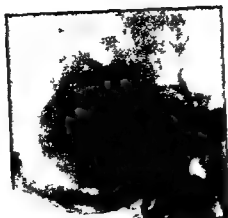


Fig 5 Angiographie vertébrale profil - Angiome circumpédunculaire

Ils sont nettement individualisés relativement limités situés à la partie distale des vaisseaux. Leurs pédicules artériels nourriciers et leurs pédicules veineux éfferents sont peu nombreux et peuvent être précisés sur les angiographies. Dans ce cas la tactique opératoire peut prévoir avant l'intervention l'approche de la malformation, la recherche des pédicules artériels et la préservation d'au moins un pédicule veineux important avant l'exérèse complète.

Ces conditions expliquent les possibilités chirurgicales et les bons résultats obtenus. Dans notre série 6 malades présentaient une telle localisation, 3 fois un traitement chirurgical a été proposé et réalisé avec succès (exérèse totale 2 fois, ligatures vasculaires et évacuation d'un hématome 1 fois).

2 Les angiomes du tronc cérébral sont moins bien individualisés et leur analyse est difficile en raison des superpositions vasculaires et osseuses qui les masquent sur les diverses incidences.

En effet certains angiomes du tronc cérébral intéressent également les hémisphères cérébelleux. Ils apparaissent comme une vaste nappe floconneuse recouvrant tout ou partie de la fosse cérébrale postérieure et se projettent en surimpression sur les pédicules qui sont difficiles à systématiser du fait de la dilution du produit de contraste et de son passage extrêmement rapide dans la lésion (Fig 4).

D'autres peuvent également s'étendre à travers le trou occipital sur les premiers segments de la moelle cervicale sans hiatus entre les deux malformations.

Les angiomes circumpédunculaires enfin représentent une forme parti-

culière des angiomes du tronc cérébral, bien que très limités, ils sont aussi difficiles à analyser, ils apparaissent mélangés au fouillis vasculaire et aux lésions des branches artérielles de cette région, intriquant la choroïdienne postérieure, la cérébelleuse supérieure et la cérébrale postérieure, nous en rapportons 3 cas (Fig 5), dont l'un fut opéré avec succès.

L'angiographie vertébrale peut montrer également un refoulement des branches artérielles au pourtour de la lésion, signant l'existence d'un hématome intraparenchymateux associé, cet aspect n'est d'ailleurs bien individualisé que dans le cas des angiomes localisés aux hémisphères cérébraux. Inversement en effet il est difficile d'affirmer sur l'angiographie vertébrale l'existence d'un tel hématome dans le cas d'angiome du tronc cérébral étendu au cervelet, ou la fosse cérébrale postérieure entière paraît recouverte d'un réseau vasculaire inextricable.

En pratique, le choix des investigations neuroradiologiques dans les angiomes de la fosse cérébrale postérieure est guidé par les caractères de leurs manifestations cliniques qui peut revêtir trois aspects essentiels : une hémorragie méningée (7 fois dans notre série), une néoformation de la fosse cérébrale postérieure (2 fois) et un syndrome vasculaire du tronc cérébral (2 fois).

La constatation d'une hémorragie méningée aboutira à un bilan vasculaire cérébral et en particulier à une angiographie vertébrale soit pratiquée à titre systématique, en l'absence de signes cliniques de localisation, soit justifiée par l'existence d'un tableau neurologique évocateur ou d'une image suspecte apparaissant déjà sur les angiographies carotidiennes.

Dans la seconde éventualité, la scène clinique évoque une néoformation de la fosse cérébrale postérieure, dont rien ne permet de suspecter l'origine vasculaire malformative. L'installation progressive ou rapide d'une hypertension intracrânienne avec œdème du fond d'œil associée à un syndrome cérébelleux conduisent à une ventriculographie. Les anomalies alors constatées — soit liées à la malformation elle-même, soit liées à un hématome intracérébelleux — font poser une indication opératoire qui redressera le diagnostic.

Enfin, en présence d'un syndrome du tronc cérébral (syndrome de Parinaud ou syndrome alterne), l'existence d'un souffle intracrânien, la survenue d'un épisode méningé ou le caractère fluctuant de l'évolution pourront faire suspecter le diagnostic dont on demandera en définitive la confirmation à l'angiographie vertébrale.

RÉSUMÉ

La connaissance des angiomes sous tentoriels repose essentiellement sur le bilan angiographique : angiographie carotidienne permettant souvent de soupçonner la malformation et surtout angiographie vertébrale. Celle-ci individualise des aspects anatomiques opposés

Les angiomes limités au cervelet dont l'angiographie détaille la disposition rendant possible une chirurgie d'exérèse. Les angiomes du tronc sont d'étude plus difficile surtout lorsqu'ils intéressent toute la fosse postérieure. Excepté l'angiome circumpédonculaire bien localisé leur étendue interdit toute tentative chirurgicale.

SUMMARY

Our knowledge concerning the subtentorial angiomas depends essentially on the angiographic findings. Carotid angiography and especially vertebral angiography often permits us to suspect the presence of this malformation. The latter examination can individualize opposed anatomical aspects. Angiomas restricted to the cerebellum whose limits can be defined by angiography are thus rendered accessible for surgical excision. Angiomas of the stem are more difficult to study especially when they involve the entire posterior fossa. Except in the case of well localized circumpeduncular angiomas their extent forbids all attempts at tentative surgery.

ZUSAMMENFASSUNG

Unsere Kenntnis der subtentorialen Angiome beruht im wesentlichen auf angiographischen Befunden. Die Gegenwart solcher Angiome kann meistens nach der Carotisangiographie besonders aber nach der Vertebralangiographie vermutet werden. Die letztere Untersuchung besonders kann die genaue Lokalisierung benachbarter anatomischer Gebiete klären. Es wird somit möglich Angiome die nur das Kleinhirn befallen zu erkennen und der chirurgischen Behandlung zugänglich zu machen. Es ist jedoch schwieriger die Angiome des Hirnstammes zu analysieren besonders wenn diese die ganze hintere Schädelgrube befallen. Mit Ausnahme der Angiome die wohl abgegrenzt sich nur um den Hirnstamm ausbreiten ist die Chirurgie durchaus kontraindiziert.

BIBLIOGRAPHIE

- CASTELLANO F, TROISI F et VITALI A. Aneurismi arteriovenosi intracraniei del sistema vertebro-basilar. *Minerva neurochir* 63 (1962) 103.
- CAMINELLO V J and SACHS E. Arteriovenous malformations of the posterior fossa. *J Neurosurg* 19 (1962) 607.
- CLÉMENT R, GERVEAUX J, COMBES-HAMELLE A, PERTUISSET M et PETRANCA C. Anévrysmes artério-veineux de l'ampoule de Galien chez nourrisson. Leur rôle dans l'hydrocéphalie non communicante. *Presse med* 62 (1954) 658.
- COHEN M and KRISTIANSEN K. Arteriovenous malformations of the great vein of Galien. *Neurology* 4 (1954) 124.
- COLMELLA F. Considerazioni su 96 casi patologici angiografia del sistema vertebro-basilar. *Chirurgia (Milano)* 8 (1953) 750.
- CRAWFORD J W and RUSSEL D J. Cryptic arteriovenous and venous hematomas of the brain. *J Neurol Neurosurg Psychiat* 19 (1956) 1.
- DANDY W E. Arteriovenous aneurysms of the brain. *Arch Surg* 17 (1928) 190.
- FRENCH L A and PEYTON W T. Vascular malformations in the region of the great vein of Galien. *J Neurosurg* 2 (1954) 488.

- GALIBERT P. et VEDRENNE C. I. Les anévrysmes artério-veineux de la fosse postérieure. Rapport Soc. Neurochir. Langue Franç., Lyon 1963.
- CINSON J. B., TAYLOR A. R. and RICHARDSON A. E. Congenital arteriovenous fistula with an aneurysm of the great cerebral vein and hydrocephalus treated surgically. *J. Neurol. Neurosurg. Psychiat.* 22 (1959) 224.
- CINSON R. M. and DA ROSSA MELO A. N. Angiographic finding of an aneurysm and arterio-venous malformation in the posterior cranial fossa in a case of subarachnoid haemorrhage. *J. Neurol. Neurosurg. Psychiat.* 23 (1960), 237.
- GIRARD P., BONNANOLLE G., ROBERT J. et LAPRAS C. A propos d'une observation d'angiome à court circuit artério-veineux de la carotide pédonculaire. *Lyon méd.* 47 (1958) 187.
- HOWL R. D. Arteriovenous aneurysm of the posterior fossa. *Acta radiol.* 40 (1953) 96.
- HOUDART R. et LI BISMARAIS Y. Les anévrysmes artério-veineux des hémisphères cérébraux. Masson Edit., Paris 1963.
- IINDEREN I. Percutaneous angiography of the vertebral artery. *Acta radiol.* 33 (1950) 389.
- McKISSOCK W., RICHARDSON A. and WALSH I. Spontaneous cerebellar haemorrhage. A study of 34 consecutive cases treated surgically. *Brain* 83 (1960) 1.
- MARCOIS G., ODOM G. I., WOODHALL B. and BLOOR B. M. The role of small angiomatous malformations in the production of intracerebellar haematoma. *J. Neurosurg.* 8 (1951) 564.
- MORILO G. Angiomi della fossa posteriore. *Comm. VIIIème Congrès Soc. It. Neurochir.* Rome 1960.
- NAYRAC L., LAINE M., FONTAN M., DELANDSIER J. M., MME DELANDSIER et GALIBERT P. Étude clinique et thérapeutique de 2 volumineux anévrysmes artério-veineux englobant le système de Galien. *Neurochirurgie* 2 (1956) 85.
- ODOM C. I., TINDALL G. T. and DUKES H. T. Cerebellar haematoma caused by angiomatous malformations. *J. Neurosurg.* 18 (1961) 777.
- OLIVECRONA H. and LADENHEIM J. Congenital arteriovenous aneurysms of the carotid and vertebral arterial systems. Springer Verlag, Berlin 1957.
- POITEN J. L. Vascular surgery of the posterior fossa. *Clin. Neurosurg.* 6 (1959) 199.
- VERBITZ H. Arteriovenous aneurysms of the posterior fossa. Analysis of six cases. *Acta neurochir.* 9 (1961) 171.
- WERTHEIMER I., ALLECRE G., LAPRAS C. et NACQUETON I. A propos de quelques aspects neurochirurgicaux de la pathologie vasculaire cérébrale chez l'enfant. *Neurochirurgie* 4 (1961) 120.
- , TUSINI G., LAPRAS C. et THIERRY A. A propos de huit observations cliniques d'angiomes de la fosse cérébrale postérieure. *Comm. VIIIème Congrès Langue Franç. Soc. Neurochir.* Lyon 1963 (à paraître).
- WICKHAM I. Angiographic examination of intracranial arteriovenous aneurysms. *Acta radiol.* 34 (1950) 385.

CONSIDERATIONS SUR LES ASPECTS ANGIOGRAPHIQUES DES ABCÈS DU CERVEAU

par

J LECUIRE P BUFFARD A GOUTELLE A THIERRY J P DECHACHE et
J KOFMAN

Le caractère clinique parfois trompeur des abcès du cerveau ne permet pas toujours d'évoquer d'emblée un diagnostic dont l'établissement ne souffre pourtant aucun retard. L'évolution de l'abcès peut être marquée comme l'ont souligné BOYVAL & DESCURS par des aggravations rapides et imprévues. Il semble donc essentiel de faire le diagnostic d'abcès sur l'artériographie afin d'affirmer la notion d'urgence. Nous avons repris le dossier radiologique de 45 abcès du cerveau pour étudier les critères angiographiques de l'affection.

L'image en cocarde ou en anneau décrite par WICKBOM en 1948 puis par HEEP en 1949 nous a paru présenter un intérêt particulier. Elle nécessite un examen minutieux sur les clichés recs en utilisant un spot lumineux.

Il s'agit d'une image ronde ou oblongue formant un anneau peu dense parfois interrompu qui entoure une plage imprégnée de façon moins marquée. L'examen à la loupe révèle une imprégnation fine et régulière constituée de fines stries concentriques. La plupart des auteurs ont insisté sur le caractère fugace de son apparition au cours de l'artériographie. L'utilisation du sériographe montre qu'une partie de la circonférence commence à être visible au

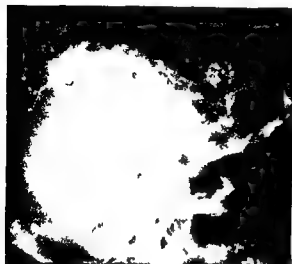


Fig 1 Arteriographie carotidienne Métériste cérébrale Image en anneau



Fig 2 Artériographie carotidienne Effoulement des veines corticales dans un cas d'abcès frontal

temps artériel. L'image apparaît cependant avec le maximum de netteté au premier temps veineux, précoce ou persiste encore un fin réseau capillaire.

Cet aspect est différent des images en anneau que donnent certaines métastases nécrosées en leur centre. Celles-ci comportent volontiers de petites dilatactions veineuses périphériques (Fig 1).

De même, dans certains gliomes du type V de WICKBOM, il est possible de rencontrer des images en anneau. Une étude soignée permet de retrouver souvent un ou plusieurs shunts artério-veineux.

L'image en cocarde nous paraît donc assez pathognomonique d'un abcès siégeant en plein parenchyme. Elle semble correspondre nullement à l'abcès lui-même. Elle a donné lieu à diverses interprétations pathogéniques résumées dans la thèse de LEMAN. Elle résulterait de l'imprégnation de capillaires tissés en dehors de la coque vasculaire de l'abcès. LOMBARDI et coll puis COLUMELLA en 1959 ont tenté de donner une valeur anatomopathologique à cette image qui traduirait une phase d'enkystement associée à une réaction périfocale.

Sur le plan pratique, cette image se rencontre également dans les cas aigus et dans les cas chroniques. Nous l'avons observée dans des cas, où n'existe pas de coque et où le pus se trouvait dans une cavité constituée au sein d'un ramollissement cérébral. Son existence paraît donc due à la présence de pus franc, elle pourrait donc apparaître immédiatement après la phase d'encéphalite pré-suppurative.

Cette image n'est cependant pas constante, WEBER en 1960, ne trouve que 8 images en cocarde sur 51 observations d'abcès. BONNAL & DESCUNS ne la

mentionnent que 22 fois sur 212 cas. Dans notre série, nous la retrouvons dans 48 % des cas ce qui constitue donc un pourcentage plus élevé.

En l'absence de cet aspect caractéristique, l'angiographie cérébrale révèle une lésion expansive entraînant d'importants déplacements vasculaires comme l'avaient décrit pour la première fois MOVIZ & LOFF en 1934.

a) Les déplacements des gros troncs artériels sont particulièrement nets. Les segments artériels étirés contrastent par leur aspect rigide avec les vaisseaux des régions saines qui ont conservé leur flexuosité normale.

b) Le déplacement veineux intéresse non seulement les veines profondes mais également les veines corticales qui paraissent volontiers tendues sur une surface sphérique (Fig. 2).

c) Dans l'écartement de ces vaisseaux, une zone avasculaire est visible, surtout au temps capillaire.

Quant aux aspects particuliers des abcès fronto-sinusiens, lorsque l'abcès est dû à une infection des sinus de la face, nous avons rencontré un aspect assez évocateur qui réalise sur le temps artériel une image en patte de crabe de la région frontale. Au temps capillaire-veineux, il s'agit d'une image avasculaire importante de la partie inférieure du lobe frontal avec bascule en arrière de l'angle veineux. Cette image particulière semble correspondre à une suppuration de proche en proche qui refoule le lobe préfrontal tout en le détruisant.

Enfin, contrairement à certains auteurs, nous n'avons jamais rencontré les images de thrombose artérielle décrites par POUYANNE, BOYVAL, DESGUNS et DUPLAY, ont d'ailleurs fait la même constatation.

En conclusion, si le stade d'encéphalite pré-suppurative semble dépourvu de traduction radiologique, l'abcès constitué déforme généralement l'angiogramme, l'artériographie carotidienne permet le plus souvent une localisation régionale précise, surtout s'il existe la caractéristique image en cocarde. La concordance entre les données anatomiques et les aspects radiologiques n'est cependant pas toujours parfaite.

Un premier examen ne permet pas toujours d'affirmer la lésion et de poser une indication opératoire. Il faut savoir dans ce cadre pathologique répéter l'artériographie.

En outre, BUGH & RACK ont décrit en 1962 trois cas où l'artériographie n'apporta aucun renseignement sur un abcès centro-hémisphérique. Ce fut également le fait d'un de nos malades.

Enfin, devant un aspect de déplacement vasculaire sans image en cocarde, il est difficile d'apprécier les anomalies dues à l'abcès lui-même, et les modifications qu'entraînent les réactions œdémateuses périfocales.

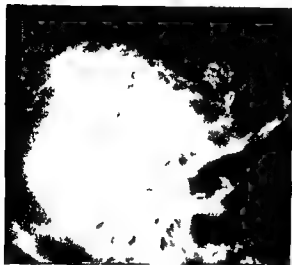


Fig 1 Arteriographie carotidienne Métastase cérébrale Image en anneau



Fig 2 Artériographie carotidienne Refoulement des veines corticales dans un cas d'abcès frontal

temps artériel L'image apparaît cependant avec le maximum de netteté au premier temps veineux précoce ou persiste encore un fin réseau capillaire

Cet aspect est différent des images en anneau que donnent certaines métastases nécrotiques en leur centre. Celles-ci comportent volontiers de petites dilatactions veineuses périphériques (Fig 1)

De même, dans certains gliomes du type V de WICKBOM, il est possible de rencontrer des images en anneau. Une étude soignée permet de retrouver souvent un ou plusieurs shunts artério-veineux

L'image en coquille nous paraît donc assez pathognomonique d'un abcès siégeant en plein parenchyme. Elle semble correspondre nullement à l'abcès lui-même. Elle a donné lieu à diverses interprétations pathogéniques résumées dans la thèse de LEMAN: elle résulterait de l'impregnation de capillaires tissés en dehors de la coque vasculaire de l'abcès. LOMBARDI et coll puis COLUMELLA en 1959 ont tenté de donner une valeur anatomopathologique à cette image qui traduirait une phase d'enkystement associée à une réaction périfocale.

Sur le plan pratique, cette image se rencontre également dans les cas aigus et dans les cas chroniques. Nous l'avons observée dans des cas où n'existait pas de coque et où le pus se trouvait dans une cavité constituée au sein d'un ramollissement cérébral. Son existence paraît donc due à la présence de pus franc; elle pourrait donc apparaître immédiatement après la phase d'encephalite pré-suppurative.

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- HEEP W Die Darstellung von Hirnabszessen im Phlebogram Zbl Neurochir 9 (1949) 2
- LECLURE J et LEVY A Ventriculographie dans l'abcès du cerveau 19 observations Rev neurol 81 (1949) 395
- LEMAN P L'angiographie carotidienne dans les abcès du cerveau Thèse Médecine 117 Bordeaux 1953
- LIMA P A Cerebral angiography Oxford Univ Press London 1950
- LOMBARDI G and MIGLIAVACCA F Radiological considerations on cerebral abscesses Minerva Neurochir 3 (1959) 117
- LUDBERG N Diagnostik und Behandlung der Hirnabszesse Acta Oto-Laryng 29 (1941) 36
- MANSUY L Quelques problèmes de diagnostic et de traitement des abcès cérébraux Lyon Chir 52 (1956) 335
- MONTE E et LOFF R L'épreuve angiographique dans le cas d'abcès du cerveau Bordeaux Chir 1 (1934) 1
- PENNYACKER J B and CANTHORNE T Discussion on diagnosis and treatment of cerebral abscess Proc roy Soc Med 38 (1945) 431
- POLYANNE L, POUYANNE H, ARNE L et LEMAN P L'angiographie cérébrale dans les abcès du cerveau (à propos de 6 observations) Rev neurol 85 (1951) 477
- SCHEER P H Cerebral angiography in abscess of the temporal lobe Brit J Surg 39 (1951) 156
- WEBER G L'angiographie dans le diagnostic des abcès cérébraux Neuro-chirurgie 6 (1960) 317
- WERTHEIMER P, LECLURE J, LAPRAS C et TUSINI C Les abcès encéphaliques chez l'enfant à propos de 16 observations Neuro-chirurgie 6 (1960) 373
- WICKBOM I Angiography of the carotid artery Acta radiol Suppl 72 Stockholm 1948

RÉSUMÉ

L'étude angiographique de 45 abcès du cerveau a permis de préciser l'importance des images en cocarde décrites par Wickham. Cet aspect visible dès le début du temps veineux parait pathognomonique de l'abcès et indique la localisation avec une précision extrême. Il doit néanmoins être distingué d'images voisines rencontrées dans les gliomes ou les métastases nécrotiques. En son absence (52 % des cas) l'abcès se manifeste sur l'artériographie comme les autres lésions expansives.

SUMMARY

An angiographic study of 45 abscesses of the brain revealed the importance of the cockade like appearance described by Wickham. This appearance which is visible from the beginning of the venous phase seems to be pathognomonic of the abscess and indicates its site with great precision. It should however not be confused with similar appearances encountered in gliomas or in necrotic metastases. In its absence (52 % of cases) the abscess gives the same manifestations at angiography as other expanding lesions.

ZUSAMMENFASSUNG

Die angiographische Untersuchung von 45 Gehirnanzessen bestätigte die Bedeutung des von Wickham beschriebenen Kokardezeichens. Dieses Zeichen kann von der venösen Frühphase an gut erkannt werden und kann als pathognomonisch für Gehirnanzess betrachtet werden. Es führt genauen Lokalisation des Krankheitsherdes. Man muss sich jedoch vor Verwechslung mit ähnlichen Erscheinungen bei Gliomen und nekrotischen Metastasen hüten. Das Zeichen fehlt in 52 % der Fälle und man kann demgemäss lediglich einen raumbeschränkenden Prozess diagnostizieren.

BIBLIOGRAPHIE

- BAGGIORE P. et BERTINI I. Possibilità angiografiche di diagnosi e di prognosi negli ascessi cerebrali. *Minerva Neurochir.* 12 (1958) 158.
- BARRE C. Aspect actuel des abcès du cerveau d'origine otitique et sinusienne. A propos de 8 cas. Thèse Médecine 626 Paris 1963.
- BLAIR A. S. and RACK P. M. H. Carotid angiography and cerebral abscess. *J. Neurosurg.* 19 (1962) 483.
- BONNAL J., DESCUNES P. et DUPLAT J. Les abcès encéphaliques à l'ère des antibiotiques. Etude statistique de 547 observations. Masson Edit. Paris 1961.
- CHICHIERO V. L. L'angiographie dans les suppurations encéphaliques otorhinogènes. Rapport du XVII Congrès d'Oto Neuro Ophthalm. Lisbonne 1952. Institut Pasteur Edit. Lisbonne 1952 79.
- COLUMELLA F., DELZANO C. M. and NICOLA G. C. A propos of the angiographical circles in cerebral abscesses. *Minerva Neurochir.* 3 (1959) 119.
- GABRITUS H. F., FROVIG A. G. and KRISTIANSEN K. Percutaneous cerebral angiography and cerebral abscess. *Arch. Neurol. Psychiat.* 61 (1949) 352.
- GÉRAUD J., LAZORTHES G., PICASSOU J. et ANDRUE H. L'angiographie cérébrale. *J. Radiol. Électrol.* 31 (1950) 141.

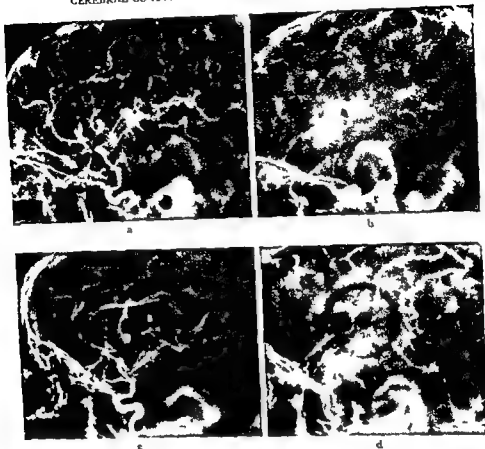


Fig 1 Patient with a temporal lobe hematoma. a) Spasm of supracallosal portion of internal carotid artery (near arrow). Origin of middle cerebral artery elevated and retrodisplaced (two arrows). Displacement of branches of middle cerebral artery (A) suggesting an intracerebral mass. b) Abnormal collection of veins in the venous phase (arrow). c) and d) Postoperative arteriography. No spasm. Branches of middle cerebral artery are now normal except for a local area of avascularity due to absence of filling of a single ascending frontal branch of middle cerebral artery (B) which may be secondary to previous surgery. The venous phase is normal.

were an additional two films at one/sec. The contrast material used was Hypaque 50%.

Clinical material. There were 39 patients examined angiographically following head trauma. None of the patients analyzed had significant subdural or epidural hematomas. There were 4 patients with minimal subdural hematomas which may have been satellite hematomas. The satellite or accessory hematoma has been described by COLUMELLA et coll (1963) as secondary to a

ANGIOGRAPHIC CHANGES IN CEREBRAL CONTUSIONS AND INTRACEREBRAL HEMATOMAS

by

N E LEEDS, N D REID and I M ROSEN

We have analyzed the arteriograms performed on 39 patients with head trauma in whom a clinical diagnosis of cerebral contusion or intracerebral hematoma was suspected, but in whom no overt angiographic evidence of subdural or extradural hematoma was present. The patients with subdural or extradural hematomas were excluded, because there have been several excellent papers on this subject (CARTON 1959, CRONQVIST & KOHLER 1963, NORMAN 1956, THOMPSON 1963).

The clinical indications for the performance of angiography following head trauma are (1) failure to improve or clinical deterioration, (2) localizing signs, (3) severe headache, and (4) the presence of papilledema.

The value of angiography in these patients is underlined by the fact that there were only 2 normal bilateral carotid arteriographies and in an additional 2 patients there was only ventricular dilatation.

Technique All the angiographies were performed utilizing a bi plane rapid serial film changer. The sequence used was two films/sec for 2 sec and one film/sec for 4 sec. If an elevation of intracranial pressure was suspected, there

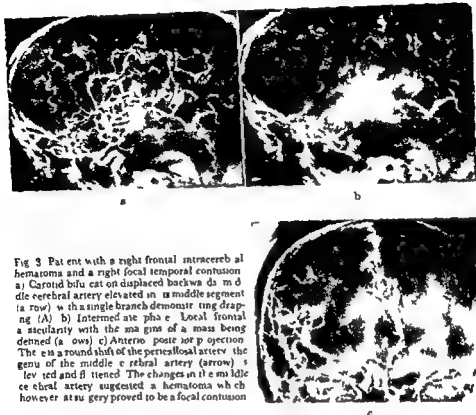


Fig 3 Patient with a right frontal intracerebral hematoma and a right focal temporal contusion a) Carotid bifurcation displaced backwards middle cerebral artery elevated in its middle segment (a row) with a single branch demonstrating draping (A) b) Intermediate phase Local frontal arteriopathy with the margins of a mass being defined (a row) c) Anteroposterior projection The arrow shows a round shift of the pericallosal artery the genu of the middle cerebral artery (arrow) is elevated and flattened The changes in the middle cerebral artery suggested a hematoma which however at surgery proved to be a focal contusion

in a local or a diffuse area. Abnormal circulation was found in 2 cases (Fig 1) and local veins appeared early in one case. The occurrence of an abnormal circulation or early filling veins in these patients is due to a local hyperemia secondary to focal cerebral hypoxia that may develop following head trauma (Pitts et coll 1964). The local increase in cerebral blood flow may lead to the shunting of blood with the appearance of early filling veins or an abnormal circulation.

The local changes in circulatory dynamics will not aid in distinguishing a cerebral contusion from an intracerebral hematoma but will aid in accurately localizing the lesion.

Arterial spasm. The occurrence of arterial spasm following head trauma was first described by Loxton in 1936. Arterial spasm may be manifested by a localized (Fig 1 a and c) or diffuse (Fig 2) decrease in the calibre of the arterial lumen. The arterial narrowing tends to be smooth and circumferential. There



Fig. 2 Patient with extensive cerebral contusions bilaterally. a) and b) Extensive spasm of supraclinoid portion of internal carotid artery and of proximal anterior and middle cerebral arteries (arrow). Spasm also on opposite side. Elevation of middle cerebral artery without draping. No shift of midline artery or of veins. (Left frontal fracture.)

cerebral laceration. These hematomas may be minimal, but on occasion may be significant.

There were 31 patients with contusions and 8 with intracerebral hematomas. Surgical or autopsy verification was established in 15 patients. These included 8 cerebral contusions, 6 intracerebral hematomas, and 1 post-traumatic encephalomalacia. There were 3 patients with minimal subdural hematomas, in 2 of these, there were cerebral contusions, and in 1 there was an intracerebral hematoma. A subdural hematoma may develop secondary to or coincident with any cerebral insult.

Lumbar punctures were performed in 34 of the 39 patients. Only 6 were normal. Twenty-seven patients had xanthochromic or bloody cerebrospinal fluid, while in one patient there was a traumatic lumbar puncture.

Skull films were obtained from 36 patients. Of these, 16 (44%) revealed skull fractures. Two of the fractures were depressed.

Angiographic changes

Local circulatory changes. There were 17 (44%) patients with an increase or decrease in local circulation (LEEDS & TAVERAS 1963). There was delayed filling of a local artery in 6 cases, delayed emptying in 2 cases, and local slowing in the intermediate (capillary) phase in 11 cases, or in the venous phase in 13 cases. This delay in filling in the intermediate and venous phases may occur

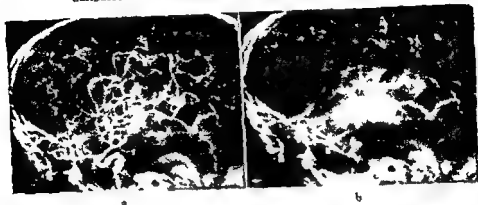


Fig 3 Patient with a right frontal intracerebral hematoma and a right focal temporal contusion. a) Cross section displaced backwards middle cerebral artery elevated in its middle segment (arrow) with a single branch demonstrating draping. (A) b) Intermediate phase. Local frontal avascularity with the margins of a mass being defined (arrows). c) Anterior-posterior projection. There is a round shift of the pericallosal artery; the genu of the middle cerebral artery (arrow) is elevated and flattened. The changes in the middle cerebral artery suggested a hematoma which however at surgery proved to be a focal contusion.



c

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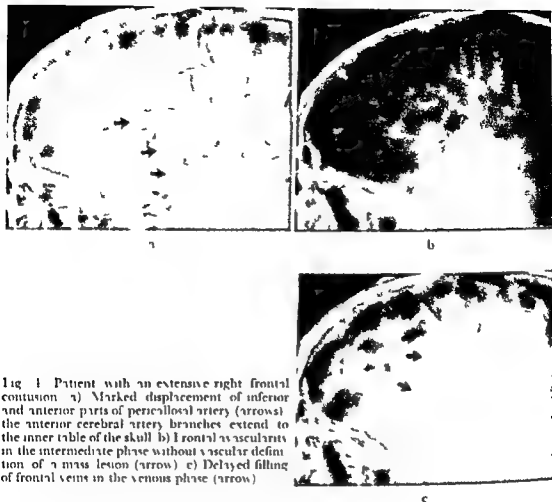


Fig. 1. Patient with an extensive right frontal contusion. a) Marked displacement of inferior and anterior parts of pericallosal artery (arrows); the anterior cerebral artery branches extend to the inner table of the skull. b) Frontal vasculature in the intermediate phase without vascular definition of a mass lesion (arrow). c) Delayed filling of frontal veins in the venous phase (arrow).

were 11 (31%) of the patients with arterial spasm. In 10 (90%) of the patients, there was bloody or xanthochromic spinal fluid. The etiology of the spasm is unknown except for the obvious relationship to the subarachnoid bleeding (COLUMELLI *et al.* 1963, FREIDENFELT & SUNDSTROM 1963). The possibility of the increased sensitivity of an artery to contrast material has been suggested as a possible cause of the spasm (RAYNOR & ROSS 1960, TAVERAS & WOOD 1964).

Differential diagnosis

To determine the best mode of therapy, it is important to distinguish between a cerebral contusion and an intracerebral hematoma. The difficulty is that contusions may be focal, and in these cases the differentiation would be impossible, except that if follow up arteriography is performed at the end of 2 to 3 weeks, there should be a return to normal in patients with a contusion



Fig 5 Patient with multiple cerebral contusions. Spasm of supracallosal portions of internal carotid artery. The carotid bifurcation, anterior cerebral artery, and the proximal and insular segment of the middle cerebral artery are elevated, spreading of the proximal ascending frontal branches of the middle cerebral artery in lateral projection. Retrodisplacement of the pericallosal artery on the lateral film.

A contusion will usually produce diffuse angiographic alterations while with a hematoma there will be focal changes (LINDGREN 1954).

Multiple angiographic lesions will usually indicate a cerebral contusion but there are patients with one area with an intracerebral hematoma and other areas of cerebral contusion (Fig 3).

A frontal contusion (Fig 4) may have an appearance similar to an intracerebral hematoma except that in the hematoma (Fig 3c) a circumferential artery or vein may be observed defining the margins of the mass lesion.

In the temporal lobe the angiographic changes that distinguish a mass from a contusion are the draping sign (Fig 1a and c) (CHASE & TAVERAS 1963) or the arterial bowing that may define the margins of the mass, but a focal contusion may produce similar changes (Fig 3a). Contusions usually produce diffuse changes (Fig 5).

Follow up arteriograms are important to evaluate any interval angiographic changes that may occur (resolution, progression or no change). There were 2 patients with angiographic evidence of resolution after an interval of 3 and 6 weeks and these were considered to have contusions because of the resolution after a relatively short interval (Fig 6). There was a patient in whom the follow up arteriogram demonstrated no change after 12 days. At surgery this was a contusion. Lack of change after a period of 1 to 2 weeks may be of no value in distinguishing a hematoma or contusion with post-traumatic encephalomalacia.

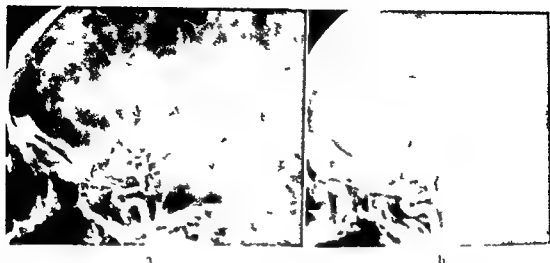


Fig. 11 Patient with a frontal opercular contusion. a) Spreading of the ascending fronto-parietal branches (A) of the middle cerebral artery. b) Four weeks later. Normal appearance of branches of the middle cerebral artery.

Generalized changes. There were 8 (21 %) patients with ventricular dilatation manifested by outward displacement of the thalamostriate vein on the anterior posterior projection in the venous phase. Two of these patients had normal sized ventricles at the first angiography with ventricular dilatation observed at a second angiography performed after an interval of 3 weeks. There were 2 (5 %) of the patients in whom the only angiographic alterations were ventricular dilatation, and only 2 (5 %) patients with normal angiograms.

SUMMARY

Angiographic changes were observed in 37 of 39 patients with severe head trauma (patients with overt evidence of subdural or extradural hematomas were excluded). Local variations in the rate of blood flow with increase or decrease in velocity were found in 17 patients. Arterial spasm was frequent. The angiographic changes that may aid in distinguishing cerebral contusions from intracerebral hematomas are discussed.

ZUSAMMENFASSUNG

Bei 37 von 39 Patienten mit schwerem Schädeltrauma wurden angiographische Veränderungen beobachtet (wobei Patienten mit klaren Anzeichen von sub und extraduralen Hämatom nicht mitgerechnet wurden). Lokale Variationen des Ausmasses der Blutzirkulation mit Beschleunigung oder Verlangsamung der Geschwindigkeit wurde bei 17 Patienten gefunden. Arterieller Spasmus war häufig vorhanden. Die angiographischen Veränderungen, die bei der Differentialdiagnose zwischen cerebralen Kontusionen und intracerebralen Hämatomen mithelfen werden besprochen.

RÉSUMÉ

Des anomalies angiographiques ont été observées dans 37 traumatismes crâniens graves sur 93 (les cas d'hématomes sous-dural ou extra-dural évidents ont été exclus). Dans 17 cas on a constaté des variations locales en plus ou en moins de la vitesse circulatoire. Le spasme artériel est fréquent. Les auteurs examinent les signes angiographiques qui peuvent aider à distinguer les contusions cérébrales des hématomes intracérébraux.

REFERENCES

- CARTON C. A. Cerebral angiography in the management of head trauma. Charles C. Thomas Springfield Illinois 1959
- CLASE V. E. and TAVERAS J. M. Temporal tumors studied by serial angiography. A review of 150 cases. *Acta radiol* 1 (1963) 275
- COLLIELLA F., DELL'ANNO G. B., GAST G. and PIAZZA G. Angiography in traumatic cerebral lacerations with special regard to some less common aspects. *Acta radiol* 1 (1963) 239
- CRONQUIST S. and HOVLER R. Angiography in epidural hematomas. *Acta radiol* 1 (1963) 47
- FREIDENFELT H. and SYDSTROM R. Local and general spasm in the internal carotid system following trauma. *Acta radiol* 1 (1963) 278
- LEEDS V. E. and TAVERAS J. M. Changes in local circulation time in frontal and parietal tumors studied by rapid serial angiography. *Acta radiol* 1 (1963) 337
- LINDGREN E. *Handbuch der Neurochirurgie* Vol II Röntgenologie. Springer Berlin Göttingen Heidelberg 1954
- LOHR W. Hirngefäßverletzungen in Arteriographischer Darstellung. I Die Arteriographie bei Compressio Cerebri. *Zbl. Chir* 63 (1936) 2466
- Hirngefäßverletzungen in Arteriographischer Darstellung. II Thrombotische Venenstoppungen und Zerebralen von Gefässen des Gehirns. *Zbl. Chir* 63 (1936) 2593
- Hirnarternienverletzungen in Arteriographischer Darstellung. *Zbl. Chir* 63 (1936) 3004
- ORRILL O. Angiographic differentiation between acute and chronic subdural and extradural hematomas. *Acta radiol* 46 (1956) 371
- PETUS F. W., HASKIN M. F., RIGGS H. E. and GROSS R. A. Tumor stain in cerebrovascular disease. *J. Neurosurg* 21 (1964) 298
- RANVIER R. B. and FOSCH C. Arteriography and vasospasm. The effects of intracarotid contrast media on vasospasm. *J. Neurosurg* 17 (1960) 1055
- TAVERAS J. M. and WOOD E. H. *Diagnostic Neuroradiology*. Williams and Wilkins Co. Baltimore Maryland 1964
- THOMSON J. L. G. Arteriography in head injuries. *Clin. Radiol* 14 (1963) 339



FIG. 6 Patient with a frontal opercular contusion. a) Spreading of the ascending fronto-parietal branches (A) of the middle cerebral artery. b) Four weeks later. Normal appearance of branches of the middle cerebral artery.

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REFERENCES

- CARTON C A Cerebral angiography in the management of head trauma Charles C Thomas Springfield Illinois 1959
- CHASE N E and TAVERAS J M Temporal tumors studied by serial angiography A review of 150 cases Acta radiol 1 (1963) 225
- COLLIMELLA F DELZANNO G ■ GAIST G and PIAZZA G Angiography in traumatic cerebral lacerations with special regard to some less common aspects Acta radiol 1 (1963) 239
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- FREIDENFELT H and SUNDSTROM R Local and general spasm in the internal carotid system following trauma Acta radiol 1 (1963) 278
- LEEDS N E and TAVERAS J M Changes in local circulation time in frontal and parietal tumors studied by rapid serial angiography Acta radiol 1 (1963) 332
- LINDGREN E Handbuch der Neurochirurgie Vol II Röntgenologie Springer Berlin Göttingen Heidelberg 1954
- LOHR W Hirngefäßverletzungen in Arteriographischer Darstellung I Die Arteriographie bei Compressio Cerebri Zbl Chir 63 (1936) 2466
- Hirngefäßverletzungen in Arteriographischer Darstellung II Thrombotische Verstopfungen und Zerreissungen von Gefässen des Gehirns Zbl Chir 63 (1936) 2593
- Hirnarterienverletzungen in Arteriographischer Darstellung Zbl Chir 63 (1936) 3004
- NORMAN D An i o r a p h i c differentiation between acute and chronic subdural and extradural hematomas Acta radiol 46 (1956) 371
- PITTS F W HASKIN M E RIGGS H E and GROFF R A Tumor stain in cerebrovascular disease J Neurosurg 21 (1964) 298
- RAYNOR R B and ROSS G Arteriography and vasospasm The effects of intracarotid contrast media on vasospasm J Neurosurg 17 (1960) 1055
- TAVERAS J M and WOOD E H Diagnostic Neuroradiology Williams and Wilkins Co Baltimore Maryland 1964
- THOMPSON J L G Arteriography in head injuries Clin Radiol 14 (1963) 339

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THREE POSITIONAL PANCLRVICAL ANGIOGRAPHY

by

EUGENI V IESLIF, GEORGE J ALKER JR and LOUIS BAIAN

A multitude of angiographic approaches to the problem of cerebrovascular disease are currently in vogue (1, 2, 5, 7, 9, 10, 11, 12, 13, 15, 16), most of them involving two or more arterial punctures, many not allowing demonstration of the aortic arch, and others not documenting the relationship of positional change to arterial narrowing. We favor the examination of the surgically accessible arterial tree from the level of the aortic arch to the base of the skull by means of a single procedure, namely, aortic arch injection. As an initial approach this allows one to determine the need for further investigation.

In brief, our technique involves the percutaneous introduction of a No. 8 teflon catheter into the ascending aorta via a femoral artery under local anesthesia. Continuous EKG monitoring is done. Three injections of 50 ml each of Hypaque 90% are made, firstly with the head and neck straight but hyperextended, and then, still with extension, rotated to the left and to the right. An effort is made to simulate any position specifically known to provoke symptoms. Biplane studies were discarded as generally unrewarding.

We prefer injection into the aorta because

1 This represents a single procedure which enables demonstration of all the surgically accessible arteries involved in cerebral blood flow, the difficulty in



Fig 1 Case 1 Normal three positional study

clinically distinguishing carotid from vertebral from diffuse cerebrovascular disease (3, 4, 14) makes an examination of the entire vascular tree essential

2 The injection at this level allows one freely to utilize position changes to evaluate their effect on arterial lumen size

3 It permits multiple views of the great vessels

4 The procedure is better tolerated than direct injection of the carotid vertebral or brachial arteries

We prefer the femoral approach over use of one of the arteries of the arm because (1) the larger caliber artery results in fewer instances of vascular spasm or occlusion at or distal to the site of puncture (2) this is an easy puncture with a relatively low failure and complication rate

The axillary artery (7, 10) however offers a good alternative in instances where a tortuous iliac artery or aorta cannot be catheterized from below

The three positional studies in addition to eliminating overlapping of some

THREE POSITIONAL PANCERVICAL ANGIOGRAPHY

by

FUGENE V LLSLIL, GEORGE J ALKER JR and LOUIS BAKAY

A multitude of angiographic approaches to the problem of cerebrovascular disease are currently in vogue (1, 2, 5, 7, 9, 10, 11, 12, 13, 15, 16), most of them involving two or more arterial punctures, many not allowing demonstration of the aortic arch, and others not documenting the relationship of positional change to arterial narrowing. We favor the examination of the surgically accessible arterial tree from the level of the aortic arch to the base of the skull by means of a single procedure, namely, aortic arch injection. As an initial approach, this allows one to determine the need for further investigation.

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Fig. 2 Case 2. Marked stenosis of origin of left internal and external carotid arteries. The three views show the lesion to best advantage.



Fig. 3 Case 3. Pre-operative angiography. The narrowing of the right vertebral artery by a large uncus vertebral spur at C6 is accentuated by rotation of the head to the right; it disappears on rotation to the left. Buckling of the left internal carotid artery occurs on rotation of the head to the left.



Fig. 4 Case 3 Post-operative angiography 8 months after anterior fusion at C 5-6. Normal caliber of right vertebral artery in all three positions. Buckling of the left internal carotid artery does not occur this time when neck was extended rather than flexed.

of the vessels also permit demonstration of partial or complete temporary occlusion of one or both vertebral arteries by uncou vertebral or apophyseal joint spurs. Buckling of the carotid and vertebral arteries may also be demonstrated by this maneuver.

This technique is of course no panacea and it has certain shortcomings namely:

- 1 The demonstration of the intracranial vessels by this method is not wholly diagnostic because of relatively poor concentration of the contrast medium and considerable overlap of vessels. This can be overcome by selective catheterization (15) or direct percutaneous injection into the appropriate artery. In any event normal neck vessels by no means exclude the possibility of intracranial disease.

- 2 Buckling is best demonstrated by using flexion while compression of vertebral arteries by uncou vertebral spurs is best demonstrated by extension of the neck. This would require at least two further views.

- 3 Ante and post mortem angiographic studies indicate that clinical significance need not necessarily be attached to any specific angiographic lesion in the neck (3-14). Perhaps radioisotopic cerebral circulation studies will help us find the appropriate subjects for angiography.

Material. During the last few years we have carried out a total of 82 pan-cervical angiograms in 78 patients. 71 patients were catheterized successfully and there were 7 failures (9%). Aside from one subintimal introduction of a



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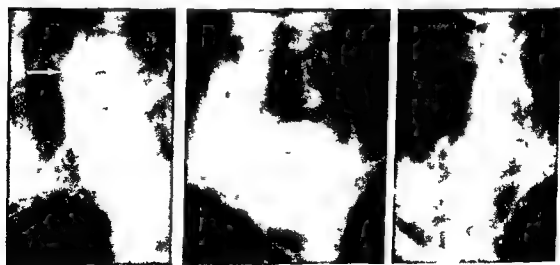


Fig. 3 Case 3. Pre-operative angiography. The narrowing of the right vertebral artery by a large uncinate vertebral spur at C. 5-6 is accentuated by rotation of the head to the right; it disappears on rotation to the left. Buckling of the left internal carotid artery occurs on rotation of the head to the left.



Fig 6 Case 4 Post-operative angiography. Only minimal indentation of the vertebral arteries at C5-6, the spurs having regressed subsequent to the fusion. Turning right and left no longer results in compression of the lumen of either vertebral artery.

the cervical portion of one or more of the carotid or vertebral arteries (in 11 patients one vessel was involved in 25 two vessels in 7 three, and in 5 four vessels)

Positional changes. These included variation in the degree of buckling of arteries and of displacement or compression of the vertebral arteries by osteophytic spurs. In 15 of those with spurs position change caused an increase in compression or displacement of an artery in 11 instances, an occlusion in 1 and a decrease in compression or displacement of 9 arteries. The caliber of an artery was sometimes decreased by rotation away from rather than toward that side, suggesting the importance of the role of apophysal joint spurring (8).

A variation in the degree of buckling with position change was encountered in 8 of 10 patients (13 vessels) who demonstrated either carotid or vertebral artery kinking when in the neutral position. In no case was the caliber reduced below 50%.

Complications. Seven complications (8.5%) occurred in this series: 1 scrotal hematoma, 1 delayed local hematoma, 2 vagovagal responses, 2 depressed ST segments in the EKG, 1 subintimal injection with back pain and shock. Three of the major complications were associated with malfunction of a particular pressure syringe.

The following illustrative cases are presented



Fig. 3. Case 4. Pre-operative angiography. Displacement of right vertebral artery at C 4—5 and compression of both vertebral arteries at C 5—6. Rotation to right effects mild compression of right vertebral artery at C 4—5 and marked compression at C 5—6. Rotation to left results in occlusion of the small left vertebral artery.

catheter, all failures occurred in patients over 65 years of age with extremely tortuous vessels. Successful catheterizations, however, have been effected up to the age of 72. Satisfactory films were obtained in 73 of the 75 successful catheterizations. (Four patients were examined twice.)

Results

As with other published series, the yield of good surgical candidates was low (3, 6). Additionally, there were two patients in this group with clinical cerebrovascular disease who had intracranial tumor. An artery was considered significantly abnormal angiographically if there was buckling, stenosis, occlusion or displacement and compression by spurs. In the vast majority the stenotic and occlusive disease was atherosclerotic in nature and involved the commonly encountered sites recorded by other authors (3, 11). The three cases of luetic or non specific arteritis showed involvement of the arch and the brachiocephalic vessels.

Occlusive disease. In 7 patients (10%) there was involvement below the origins of the right common carotid and vertebral arteries. One of these had occlusion of the origin of the left common carotid artery, this being considered an arch abnormality. The subclavian artery was involved in 9 instances and the innominate artery in one. A total of 46 patients (66.6%) had involvement of

SUMMARY

Experiences with transfemoral three positional pancervical angiography are reviewed. This in our opinion is the simplest best tolerated and most informative initial examination but it does not necessarily obviate the need for precise intra-cranial angiography. Buckling of the carotid artery is of lesser significance than vertebral artery compression by osteophytic spurs. Relief of the latter has in two of our patients been accomplished by anterior cervical fusion.

ZUSAMMENFASSUNG

Unsere Erfahrung mit der drei positionalen pancervicalen Angiographie wird erläutert. Diese stellt die einfachste am besten verträgliche und informative Untersuchung dar ohne dass sie eine genau durchgeführte intrakranielle Angiographie überflüssig macht. Deformierungen der A. carotis sind von geringerer Bedeutung als die Kompression der A. vertebralis durch Osteophyten. Letztere konnten bei zwei unserer Patienten durch eine anteriore cervicale Fusion beseitigt werden.

RÉSUMÉ

Les auteurs tirent les conclusions de leur expérience de l'angiographie pancervicale à trois positions par voie fémorale. C'est l'examen initial le plus simple le mieux toléré et le plus instructif mais elle ne dispense pas toujours d'une angiographie intracrânienne précise. Les tortuosités de la carotide ont moins d'importance que la compression de l'artère vertébrale par des becs ostéophytiques. Le traitement de cette compression a été fait chez deux des malades des auteurs par fusion cervicale antérieure.

REFERENCES

1. ANPLATZ J, RESCH J and HILAL S. A catheter approach for cerebral angiography. *Radiology* 81 (1963) 576.
2. BUNISTER D G and MURRAY LESLIE R M. Veno-angiography in cerebrovascular disease. *Brain* 86 (1963) 161.
3. BAUER R B, SHEEHAN M, WECHSLER N and MEYER J S. Arteriographic study of sites incidence and treatment of arteriosclerotic cerebrovascular lesions. *Neurology* 12 (1962) 698.
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Case 1 (normal) L. K., a 58 year old Indian male with rheumatic heart disease was investigated for possible occlusive cerebrovascular disease. His three positional pancervical angiogram (Fig. 1) was essentially normal. The origins of the vessels, the carotid bifurcations and the entirety of the surgically accessible cerebral arterial supply were demonstrated. The neutral, right and left rotation series provided multiple views, avoided overlap and elucidated any effect of position change on arterial lumen size. Serial filming, occasionally obviates overlap also, especially of the vertebral artery origin.

Case 2 (occlusive disease) G. C., a 66 year old white male with symptoms of cerebrovascular insufficiency showed stenosis of the left internal and external carotid arteries (Fig. 2). The multiple views aided in delineating the extent of the process and obviated overlapping.

Case 3 (vertebral artery compression and carotid buckling) S. W., a 42 year old colored male had complaints of dizziness and occipital headache aggravated by extension of the neck and turning of the head to the right. His pre-operative three positional study (Fig. 3) showed multiple unco-vertebral spurs indenting the vertebral arteries. Narrowing of the right vertebral artery at C 5-6 was notably accentuated by turning to the right and diminished on turning to the left. On the latter maneuver the left internal carotid buckled. The neck was somewhat flexed this time.

A second angiography (Fig. 4) 8 months following anterior fusion at C 5-6 showed marked diminution in the displacement of the right vertebral artery at this level and no compression of this artery on turning to the right. On turning to the left with the head more extended this time no buckling of the left carotid artery occurred. The patient considered himself cured.

Case 4 (vertebral artery compression and occlusion) R. G., a 47 year old white male entered hospital complaining of positionally induced dizziness and faintness. His pre-operative angiography (Fig. 5) revealed mild compression of the vertebral arteries by unco-vertebral spurs at C 5-6 and mild displacement of the right vertebral artery by spurs at C 4-5. On turning to the left the notably smaller left artery was occluded by the C5-6 spurs and the compression of the right vertebral artery disappeared. The displacement at the C 4-5 on the right was unchanged. On turning to the right there was slight compression of the right vertebral artery at C 4-5 and marked compression at C 5-6.

Six months following anterior fusion at C 5-6 a repeat angiography (Fig. 6) showed considerable diminution of the spurs. There was no compression of the vertebral arteries on turning to the right or the left.

Conclusions

There is no single angiographic approach to the study of cerebrovascular disease which answers all of our needs, but in our opinion, transforaminal three positional pancervical angiography is the single, simplest, best tolerated and most informative initial study. It does not obviate the need for precise intracranial angiography by selective catheterization (15) or direct puncture of an appropriate carotid or vertebral artery. There remains a considerable discrepancy between angiographically demonstrable lesions and symptomatically important lesions. While buckling has not been found to be of great significance in our experience, notable relief of vertebral artery compression by spurs subsequent to anterior cervical fusion has been well documented by pre and post operative three positional studies.

SUMMARY

Experiences with transfemoral three positional pancervical angiography are reviewed. This in our opinion is the simplest best tolerated and most informative initial examination but it does not necessarily obviate the need for precise intra-cranial angiography. Buckling of the carotid artery is of lesser significance than vertebral artery compression by osteophytic spurs. Relief of the latter has in two of our patients been accomplished by anterior cervical fusion.

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REFERENCES

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ARTERIOVENOUS FISTULA AFTER PERCUTANEOUS VERTEBRAL ANGIOGRAPHY

by

JACK LESTER

More than a hundred arteriovenous fistulas on the vertebral arteries most of them after penetrating injuries have previously been described (ELKIN & HARRIS 1946) but ARONSON (1961) was the first to demonstrate these fistulas arteriographically.

Although SUGAR et coll. as early as 1949 mentioned the possibility of an arteriovenous fistula as a complication of percutaneous vertebral angiography, these fistulas are apparently uncommon as only a few cases have been described in the literature since OLSON et coll. in 1963 published the first.

The three additional cases of this complication reported here suggest that it is perhaps not as rare as has been presumed. As they are to be included in a future paper (JACOBSEN et coll.) which also describes two fistulas after closed injuries they will only be summarized here.

Case reports

Case 1 Male aged 21 with a subarachnoid haemorrhage without focal neurologic symptoms or signs. Bilateral carotid angiography and percutaneous left vertebral angiography under taken by a less experienced operator revealed nothing abnormal. After this last examination the patient was troubled by tinnitus and he was re-admitted 7 months later for further investigation. This revealed a machinery murmur over the middle of the left side of the neck and

- 9 MARSHALL T. R. and LINC J. T. Direct percutaneous non catheter left and right brachial angiography. *Radiology* 80 (1963) 258
- 10 NEWTON T. H. The axillary artery approach to arteriography of the aorta and its branches. *Am J Roentgenol* 89 (1963) 275
- 11 POSTER C. M. SNODGRASS R. C. and FARIS V. A. Radiologic visualization of neck vessels in cerebrovascular insufficiency. *J Amer med Ass* 182 (1962) 126
- 12 SHEEHAN S., BAUER R. B. and MEYER J. S. Vertebral artery compression in cervical spondylosis. *Neurology* 10 (1960) 968
- 13 SHENKIN H. A., TATSUMI T. and BENTLEY D. Simplified method for total cerebral angiography. *J Amer med Ass* 182 (1962) 132
- 14 STEIN B. M., MCCORMICK W. F., RODRIGUEZ J. N. and TVERAS J. M. Radiography of atheromatous disease involving the extracranial arteries as seen at post mortem. *Acta radiol* 1 (1963) 455
- 15 WISCOTT S. L., CHANN K. Y. and STEINBERG I. Percutaneous transfemoral selective arteriography of the brachiocephalic vessels. *Am J Roentgenol* 90 (1963) 554
- 16 WHALEN R. F., HRYMAN A. and MCINTOSH H. D. Cinefluoroscopy of extracranial cerebral circulation. *Circulation* 28 (1963) 503

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Transfemoral catheterization demonstrates arteriovenous fistula on the left vertebral artery 6 months after percutaneous vertebral angiography

an arteriovenous fistula of the left vertebral artery was demonstrated by transfemoral catheterization (see accompanying illustration)

Case 2 Boy aged 14 with attacks of rotatory vertigo, tinnitus and headache. The left plantar response was extensor but no other abnormal neurologic signs were found. On suspicion of a vascular malformation, left vertebral angiography was done by a less experienced operator. The angiography revealed nothing abnormal. After this the tinnitus changed from a humming noise it turned into a rhythmic blowing sound. The remaining symptoms were unchanged. The patient was re-admitted six months later and a machinery murmur was auscultated over the left side of the neck. Transfemoral angiography revealed an arteriovenous fistula on the left vertebral artery similar to the one found in case 1.

Case 3 Woman aged 26 with a six months history of rotatory vertigo and headache. For two weeks she had had buzzing in both ears. She was admitted to another hospital and on suspicion of a space occupying, infratentorial lesion, left sided percutaneous vertebral angiography was attempted. The examination was unsuccessful as the needle could not be held in position in the artery. Sixteen ml Urografin 60° was injected but no intracranial filling was obtained. After this the tinnitus increased and a rhythmic blowing sound which had not been present before the examination could be heard on the left side of the neck. The patient was transferred to our hospital and 16 days later a transfemoral catheterization revealed an arteriovenous fistula on the left vertebral artery similar to the fistulas seen in cases 1 and 2.

Discussion

The angiographic technique used was that described by LINDGREN (1950), but a Sheldon needle with a small side aperture 2 to 3 mm from the trocar pointed tip as modified by SWANN (1958), was used instead of the ordinary short bevel needle. After the introduction of the Swann needle we considered that puncture of the vertebral artery was so simple that every neuroradiologist experienced in carotid angiography should be able to perform vertebral angiography. However on analysis of 337 examinations carried out over a period of 5 years (LESTER & KLEE) we found 8 local complications including 6 cases of brachial plexus irritation and 2 arteriovenous fistulas (cases 1 and 2). In all these 8 cases the angiography had been carried out by less experienced investigators under supervision whereas the really experienced operators who performed 45 % of all examinations had caused no local complications. It was also found that in 6 of 8 cases with local complications including the 2 cases of arteriovenous fistula the investigator had had technical difficulties. Consequently, we have now made it a rule that only experienced, qualified neuroradiologists carry out percutaneous vertebral angiography.

Arteriovenous fistulas are also seen after diagnostic puncture of other arteries for instance the two arteries most frequently used for indirect vertebral angiography the brachial (SAMUEL 1962) and the femoral (McAFEE 1957). The risk of producing a fistula of the vertebral artery is probably greater however as with that artery the puncture hole cannot be closed by digital compression.

Although an arteriovenous fistula on the vertebral artery may be a serious complication we still consider percutaneous vertebral angiography performed by a skilled and experienced operator to be a rapid and satisfactory examination.

SUMMARY

Three cases of arteriovenous fistula on the vertebral artery have been demonstrated by transfemoral catheterization. All of them developed after a previous percutaneous vertebral angiography performed by a less experienced operator under technical difficulties. Consequently only skilled experienced investigators should carry out these examinations.

ZUSAMMENFASSUNG

Mittels transfemorale Katheterisierung wurden drei Fälle von arteriovenösen Fisteln der Vertebralarterie gezeigt. Alle Fälle entwickelten sich im Anschluss an eine frühere perkutane Vertebralisangiographie, die von einem weniger erfahrenen Untersucher unter technischen Schwierigkeiten vorgenommen worden war. Es ergibt sich daraus, dass nur geschickte, erfahrene Untersucher diese Angiographie durchführen sollten.

RÉSUMÉ

Trois cas de fistule artério-veineuse de l'artère vertébrale ont été mis en évidence par cathétérisme transfémoral. Tous sont apparus après une angiographie vertébrale percutanée faite par un opérateur moins expérimenté au milieu de difficultés techniques. Il faudrait donc que ces examens ne soient faits que par des opérateurs adroits et expérimentés.

REFERENCES

- ARONSON N. I. Traumatic arteriovenous fistula of the vertebral vessels. *Neurology* 11 (1961) 817.
- FAIN D. C. and HARRIS M. H. Arteriovenous aneurysm of the vertebral vessels. *Ann Surg.* 124 (1946) 934.
- JACOBSEN B., KIER A. and LESTER J. Arteriovenous aneurysm of the vertebral vessels. To be published.
- LESTER J. and ALLEN A. Complications of 337 percutaneous vertebral angiographies. *Acta neurol. scand.* 41 (1965) 301.
- LENDGREN I. Percutaneous angiography of the vertebral artery. *Acta radiol.* 33 (1950) 399.
- Another method of vertebral angiography. *Acta radiol.* 46 (1956) 257.
- McAFFEE J. G. A survey of complications of abdominal aortography. *Radiology* 68 (1957) 825.
- OLSON R. W., BAUER H. L. and SIMEN H. J. Arteriovenous fistula: a complication of vertebral angiography. *J. Neurosurg.* 20 (1963) 73.
- SAMUEL I. Percutaneous brachial catheterization. *Brit J. Radiol.* 35 (1962) 468.
- SUGAR O., HOLDEN L. B. and POWELL C. H. Vertebral angiography. *Amer. J. Roentgenol.* 61 (1949) 166.
- SWANN G. F. Vertebral angiography using the Sheldon needle and modification of it. *Brit J. Radiol.* 31 (1958) 23.

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MICROANGIOGRAPHIC CHANGES IN THE TRAUMATIZED BRAIN

by

J PEN TZE LIN N E CHASE I I KRICHEFF, F ALEU M GOLDBAUM and
W K HASS

The increased use of motorized transportation has caused a sharp rise in the incidence of acute head trauma. The treatment of serious acute head trauma is often unrewarding and the reasons for poor response to treatment in many instances are not well understood (BENDER 1960 CLARKE & ROWBOTHAM 1963 HUNTER 1960). The purpose of this investigation was to determine if any changes might be demonstrated in the microscopic vessels in the traumatized brain by microangiographic techniques. Human autopsy material was used initially but because of lack of control over the time interval between death and autopsy and an incomplete knowledge of the exact nature and amplitude of the injuring force it was decided to concentrate our work on laboratory animals which could be studied under optimal controlled conditions.

Material and methods

Albino male rats weighing 300 gm were traumatized with a specially con-

RÉSUMÉ

Trois cas de fistule artério veineuse de l'artère vertébrale ont été mis en évidence par cathétérisme transfémoral. Tous sont apparus après une angiographie vertébrale percutanée faite par un opérateur moins expérimenté au milieu de difficultés techniques. Il faudrait donc que ces examens ne soient faits que par des opérateurs adroits et expérimentés.

REFERENCES

- ARONSON N I: Intracranial arteriovenous fistula of the vertebral vessels. *Neurology* 11 (1961) 817
- ELAIN D C and HARRIS M H: Arteriovenous aneurysm of the vertebral vessels. *Ann Surg* 124 (1946) 934
- JACOBSEN H, KIFF A and LESTER J: Arteriovenous aneurysm of the vertebral vessels. To be published
- LESTER J and KIFF A: Complications of 337 percutaneous vertebral angiographies. *Acta neurol scand* 41 (1965) 301
- LINDGREN F: Percutaneous angiography of the vertebral artery. *Acta radiol* 33 (1950) 389
- Another method of vertebral angiography. *Acta radiol* 46 (1956) 257
- McAFFEE J C: A survey of complications of abdominal aortography. *Radiology* 70 (1957) 873
- OLSON R W, BAKER H L and SILVER H J: Arteriovenous fistula: a complication of vertebral angiography. *J Neurosurg*, 20 (1963) 73
- SAMUEL F: Percutaneous brachial catheterization. *Brit J Radiol* 35 (1962) 468
- SUGAR O, HOLDEN L B and POWELL C B: Vertebral angiography. *Amer J Roentgenol* 61 (1949) 166
- SWANN G I: Vertebral angiography using the Sheldon needle and modification of it. *Brit J Radiol* 31 (1958) 23



Fig 1. Normal microangiogram. *Top view*. Orderly parallel perforating vessels perpendicular to brain surface with a size of about 80 micra. *100 \times* . *Lower view*. Good demonstration of capillary beds. *200 \times* .

(1963) and by PEREZ et coll (1964) with a size of about 80 micra. There was good demonstration of the capillary bed (Fig 1). The microangiogram of the immediate post trauma group demonstrated marked diminution in the caliber of the perforating vessels to a size of less than 20 micra. There was also reduction in the capillary networks (Fig 2). The one day post trauma microangiogram showed a return toward normal in the caliber of the vessels but an increased tortuosity of those vessels. There was further diminution in the filling of the capillaries (Fig 3). A gradual return to normal appearance could be seen on the microangiograms on succeeding days. The caliber of the perforating vessels had almost returned to normal in the five days post trauma group and the filling of the capillary bed was only moderately decreased (Fig 4).

The most significant histologic feature was the presence of an edematous

platform and immobilized. The rat was brought to the third stage of anesthesia with ether and as the rat began to come out of anesthesia, a brass ball was dropped from a height of 90 cm. In this way, a sudden momentum of 2.9×10^4 dyne seconds (momentum $ft = mv$, $v = \sqrt{2gh}$) was applied to the vertex of the rat's skull. Twenty one rats were divided into seven groups. One non-traumatized group was used as a control. The other six groups were traumatized on the same day. Microangiography was performed immediately post trauma on the first group. The other groups were studied, one group for each succeeding day for five days. Prior to microangiography, the rats were anesthetized with 15 mg of Nembutal intraperitoneally. The abdominal cavity was opened and the abdominal aorta and inferior vena cava were separated. A 19 gauge blunt cannula was introduced into the abdominal aorta and the inferior vena cava was sectioned. Heparinized dextrose solution was injected into the aorta until there was a clear return from the inferior vena cava. Then a 75% microbarium suspension in a 2% gelatin solution was injected into the aorta at a constant pressure of 300 mm Hg by gravity for 20 to 30 minutes (colloid barium sulfate, particle size range from 0.1 to 0.3 micra, Bell Crude, Inc., New York). The rats were refrigerated but not frozen for 2 hours before removing the brain. The brain was fixed in a 10% buffered formalin solution for 3 days. 0.5 mm and 1.0 mm frozen sections were obtained for radiograms. The radiograms were obtained on Kodak high resolution plates using a Beryllium window x-ray tube with the exposure factors of 16 kV, 40 mA and 20 minutes exposure time at a distance of 100 cm. The plates were viewed with a microscope and photomicrographs were obtained at 25 power and 50 power magnifications. Frozen and paraffin sections were also taken for histologic examination.

Results

Clinically the typical reaction immediately post trauma was that the rats would curl up and stop breathing for about 20 seconds, the respiration gradually returned to normal over a period of about 5 minutes. By then they were completely awake, though not too active. Gross examination of the intracranial contents of the immediate and one day post trauma groups revealed obvious subdural and subarachnoid blood. The amount of subdural and subarachnoid blood decreased rapidly with time so that none could be observed on the third, fourth and fifth post trauma days.

The normal microangiogram demonstrated orderly, parallel perforating vessels perpendicular to the brain surface as described by ZEMAN & INNES



Fig. 3. Microangiogram of one day post trauma group. Top view. A return toward normal in caliber of perforating vessels but in increased tortuosity of these vessels. 100 \times . Lower view. A further diminution in filling of capillaries. 200 \times .

Discussion

The temporary respiratory depression of the rats immediately after the strike was also observed by DENNY BROWN & RUSSELL (1941). They attributed it to the stimulation of the vago glossopharyngeal system by the subthreshold blows. In this study, the severe narrowing of the perforating vessels as seen in the immediate post trauma group also might be sufficient to cause transient anemia of the brain and lead to temporary respiratory depression.

FLOREY in 1925 found that the cerebral vessels of cats reacted to mechanical, thermal, electrical and chemical stimuli by contraction and he could not demonstrate the evidence of any nervous control over the caliber of the cerebral vessels. ECHLIN (1942) also demonstrated constriction of individual pial blood vessels following mechanical and electrical stimuli, and the constriction was not dependent on neurovascular mechanism. DENNY BROWN & RUSSELL did



Fig 2 Microangiogram of immediate post trauma group *Top view* Marked diminution in caliber of perforating vessels to a size of less than 20 micra $100\times$ *Lower view* Reduction in filling of capillary networks compared with fig 1 lower view $200\times$

reaction which became more apparent one day after the traumatic injury. The edema was noted in cortex and white matter. It should be noted that rats have very little white matter and therefore fluid accumulation was more significant within the cortex. In our animals the cortical glial cells (astrocytes) exhibited marked swelling and vacuolization (Fig 5). Clear vacuoles were also noted within the white matter. The edema was much less apparent in the animals studied immediately and five days after the traumatic injury (Fig 6). It should be emphasized that in these experiments inflammatory changes associated with cellular infiltrates did not occur. Evidence of neuronal degenerative changes was not found. A number of medium sized vascular structures appeared to have very prominent clear perivascular zones which seemed to be more prominent in the animals sacrificed immediately after trauma (Fig 7).



Fig. 3 Microangiogram of one day post trauma group. Top view: A return toward normal in caliber of perforating vessels but in increased tortuosity of these vessels. 100 \times . Lower view: A further diminution in filling of capillaries. 200 \times .

Discussion

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Fig. 1. Microangiogram of five days post trauma group. *Top view.* Almost normal appearance of perforating vessels compared with fig. 1 top view 100 \times . *Lower view.* Moderately decreased capillary networks compared with fig. 1 lower view 200 \times .

not find evidence of cerebral vasospasm by direct stimulation of the vasomotor center in the medulla of dogs. Our explanation for the marked vascular narrowing as seen in the immediate post trauma group was that it was the result of mechanical stimulation and the presence of subarachnoid blood. The sudden strike on the vertex of the rat's skull induced a sudden movement of the brain within the cranial cavity which acted as a direct mechanical stimulation to the cerebral vessels and resulted in vascular narrowing. The presence of subarachnoid blood might also play a role in the development of vascular narrowing. Individual and topographic differences accounted for a wide range in the size of the vessels as noted on histologic sections. This precluded a meaningful evaluation of the presence or absence of vascular spasm. Equally dangerous would be to draw inferences from the appearance of the clear perivascular zone. The prominence of this region could be enhanced

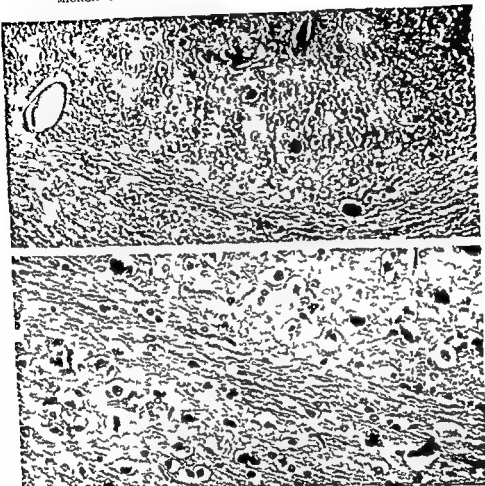


Fig 5 Histologic section of one day post trauma group. Top view: Marked swelling and vacuolization of cortical glial cells (astrocytes) and clear vacuoles within the white matter. 100 \times . Lower view: 400 \times .

by marked reduction of the vessel size, swelling of the perivascular astrocytic foot processes and shrinkage and sectioning artifacts. There was no evidence of neuronal damage in our specimens as was seen in the studies of GROOT et coll (1945), EVANS (1945) and WINDLE et coll (1944).

In the one and two days post trauma group, the cortical vessels were tortuous as seen in the microangiograms, and there was evidence of cerebral swelling in the corresponding histologic sections. These findings were consistent with the findings seen in experimental cerebral edema by PEREZ et coll



Fig 6 (Above) Histologic section of five days post trauma group showed that the edema was much less apparent 100



Fig 7 (Below) Histologic section of immediate post trauma group showed very prominent clear perivascular zones 100

ic, the tortuosity of the vessels was due to their compression by the expanding swollen brain tissues

ROCKOFF & OMMAYA'S (1964) experiment demonstrated consistent cerebral circulatory slowing following head trauma in monkeys. They attributed this to arterial spasm, cerebral edema and the development of intravascular aggre-



Fig 8 Carotid angiogram of a patient with temporal contusion demonstrated generalized spasm of anterior and middle cerebral arteries. Middle cerebral artery was elevated and displaced medially. There was no evidence of subdural hematoma.

gations of blood cells. The present study demonstrated that vascular narrowing occurred prior to the development of cerebral edema and may be due to unrelated factors.

It has been our experience and the experience of others (COLUMELLA et coll 1963, FREIDENFELT & SUNDSTROM 1963 and GREITZ & LINDGREN 1961) to see localized or generalized areas of vascular narrowing in angiograms of patients with severe head trauma (Fig 8). It now seems probable from this work that there is also extensive narrowing of the microvasculature in the patients with severe head trauma. This generalized vascular narrowing may in part be responsible for the cerebral signs accompanying head trauma and may explain the frequent lack of response to treatment. It also is important to stress the fact that the vascular narrowing occurred prior to the development of cerebral edema. This adds supporting evidence to the concept of independent and separate mechanisms for the establishment of vascular spasm and that of cerebral edema.

Acknowledgements

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- HUNTER A R Basic considerations in the conservation care of head injuries *Postgrad med J* 36 (1960) 370
- PEREZ C A HODGES III F I and MARCUS A R Microangiography in experimental cerebral edema in rats *Radiology* 82 (1964) 529
- ROCKOFF S D and OMURA A K Experimental head trauma cerebral angiographic observations in the early post traumatic period *Amer J Roentgenol* 91 (1964) 1026
- WINDLE W F GROOT III A and FOX C A Experimental structural alterations in the brain during and after concussion *Surg Gynec Obstet* 79 (1944) 561
- ZEMAN W and INNES J R M Craigie's neuroanatomy of the rat Academic Press New York and London 1963

SUMMARY

Microangiographic findings after experimental head traumas in animals are described. The immediate post trauma group demonstrated marked diminution in the caliber of the perforating vessels which occurred prior to the development of cerebral edema. The microangiographic findings were correlated with histologic findings. The possible mechanisms for the vascular spasm are discussed.

ZUSAMMENFASSUNG

Es werden mikroangiographische Befunde nach experimentellen Schädelverletzungen an Tieren beschrieben. Die posttraumatische Gruppe zeigte unmittelbar nach den Trauma eine markante Verminderung des Kalibers der perforierenden Gefäße noch bevor sich ein zerebrales Ödem entwickelte. Die mikroangiographischen werden mit den histologischen Befunden korreliert. Die möglichen Mechanismen die zum Auftreten von Gefäßspasmen führen werden besprochen.

RÉSUMÉ

Description des lésions microangiographiques cérébrales après traumatismes crâniens expérimentaux chez des animaux. On constate dans le groupe d'animaux étudié immédiatement après le traumatisme une réduction marquée du calibre des vaisseaux perforants qui apparaît avant l'œdème cérébral. Les signes microangiographiques sont confrontés avec les signes histologiques. L'auteur examine les mécanismes possibles du spasme vasculaire.

REFERENCES

- BENDER M. B. Recovery from subdural hematoma without surgery. *Mt. Sinai Hosp. J.* (N.Y.) 27 (1960) 52.
- CLARK P. R. R. and ROWBOTHAM G. F. The management of head injuries. *Postgrad. med. J.* 39 (1963) 709.
- COLUPELLA T., DELZANNO G. B., GAIST G. and PIAZZA G. Angiography in traumatic cerebral lacerations with special regard to some less common aspects. *Acta radiol.* 1 (1963) 239.
- DENNY BROWN D. and RUSSELL W. R. Experimental cerebral concussion. *Brain* 64 (1941) 93.
- FCHLIN F. A. Vasospasm and focal cerebral ischemia (An experimental study). *Arch. Neurol. Psychiat.* (Chicago) 47 (1942) 77.
- EVANS J. P. Histologic studies of the brain following head trauma. *J. Neurosurg.* 2 (1945) 306.
- FLORLY H. Microscopical observation on the circulation of the blood in the cerebral cortex. *Brain* 48 (1925) 43.
- FREIDENFELT H. and SUNDSTROM R. Local and general spasm in the internal carotid system following trauma. *Acta radiol.* 1 (1963) 278.
- GREITZ T. and LINDQVIST T. Intracranial traumatic changes. *Angiography* (Edited by Abrams H. L.) Vol. 1. Chap. 7. P. 95. Little Brown & Co. Boston Mass. 1961.
- GROAT R. A., WINDLE W. F. and MAGOWN H. W. Functional and structural changes in the monkey's brain during and after concussion. *J. Neurosurg.* 2 (1945) 26.

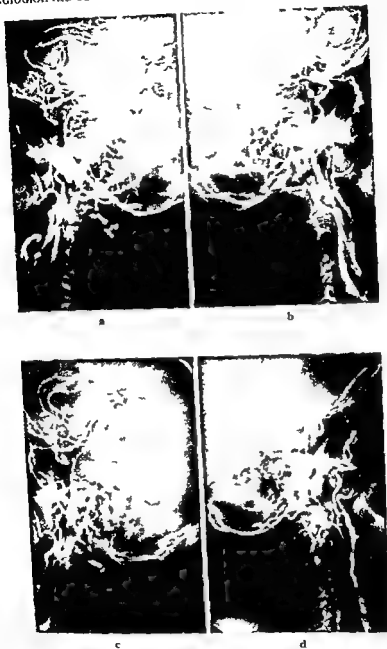


Fig 1 a) and b) B lateral carotid angiogram showing marked atherosclerotic changes in both carotid systems. No complications arose. c) and d) Repeat examination 9 months later. Occlusion of both internal carotid arteries. Hemiparesis occurred immediately after this study.

COMPLICATIONS OF ANGIOGRAPHY IN PATIENTS WITH VASCULAR AND ANAPLASTIC AND OTHER DISEASE OF THE NERVOUS SYSTEM

by

D W LINDNER and E S GURDJIAN

Cerebral angiography has acquired sudden and widespread acceptance and use in this country in the past ten years. At the present time it is mandatory for the proper evaluation of certain vascular disorders affecting the central nervous system.

Any classification of complications of angiography must of necessity be a rather complex one, since each of the various mechanisms must be considered in the light of on the one hand the many conditions for which angiography is performed and on the other hand the wide range in the ages and physical conditions of the patients being examined. A simple classification to be used in this paper utilizes four categories: (1) chemical, (2) mechanical, (3) infectious, and (4) complications arising from the condition of the patient and the disease process. Obviously, there is some overlapping and the exact placement of each and every complication into one of the above headings may be impossible, but such a consideration is valuable for discussion.

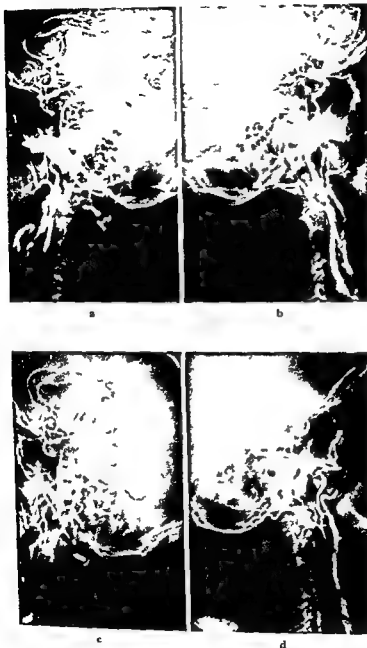


Fig 1 a) and b) Bilateral carotid angiogram showing marked atheromatous changes in both carotid systems. No complications arose. c) and d) Repeat examination 9 months later. Occlusion of both internal carotid arteries. Hemiparesis occurred immediately after this study.

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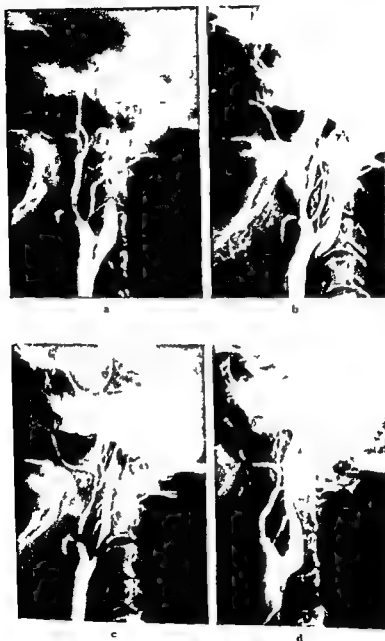


Fig 3 a) Subarachnoid injection b) Repeat injection after readjustment of needle in lumen of vessel c) Repeat injection several hours after (a) and (b) Central lateral hemiparesis had developed d) Postendarterectomy (Hemiparesis cleared after endarterectomy)

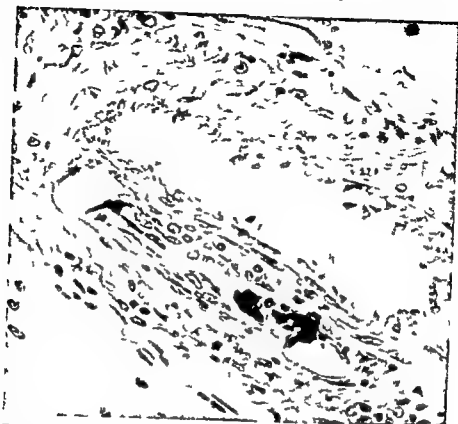


Fig 2 Asymptomatic cotton fiber embolus following carotid angiography

Causes of complications

Chemical effects The intrinsic toxicity of the contrast material may itself cause complications. It is universally accepted that Diodrast is toxic to the cerebral vessels. Prior to our use of sodium diatrizoate (Hypaque) we performed a number of animal experiments in an attempt to determine the relative specific toxicity to the cerebral vessels of Diodrast, Hypaque, Renografin, Urokon, and Thorotrast (LINDNER et coll 1958). In brief, the results of these experiments revealed that Diodrast and Urokon were extremely toxic, this being measured by the technique described by BROMAN & OLSSON (1918). Thorotrast in this particular respect was by far the least toxic of all, although it has delayed effects.

The effects of rapid injection of Hypaque into the carotid artery of a dog were usually fourfold: (1) transient apnea followed by temporary increase in respiration, (2) temporary slowing of the pulse, (3) temporary dilatation of the pupil on the side of the injection, and (4) occasional movements of the facial musculature. These effects were present to greater or lesser extent with the other contrast media (except Thorotrast). The more toxic materials



Fig 4 a) Subintimal injection. Needle was withdrawn and pressure applied over puncture site
b) Repeat injection 3 days later

vessel (Fig 3). When this is observed on the initial film, it is wise to withdraw the needle immediately and observe the patient closely for any developing neurologic deficits. Infrequently, subintimal injection can result in vessel occlusion and severe neurologic deficit. However, in a majority of cases, simple compression at the site of injection for a period of minutes may result in absorption of the contrast material and reestablishment of normal flow within the artery (Fig 4).

A delayed effect of interstitial injection with weakening of the arterial wall may result in aneurysm formation (Fig 5). The initial angiographic study with an interstitial injection at the point of the needle is seen to the left. Within three weeks, an aneurysmal dilatation developed directly at the site of the interstitial injection.

There are other mechanical effects unrelated to actual injections but directly related to manipulation by the point of a needle. We are somewhat surprised that more cases of jugular carotid fistulae have not been reported

caused seizures immediately after injection in some animals. Using the technique described by BROMAN & OLSSON, two types of changes were noted. In some animals, there was a 'regional' effect, consisting of stunning of the brain in the area bathed by the vessel injected. In other animals, there were punctate hemorrhages, usually in the region of the basal ganglia. These latter changes were frequently noted in brains not showing the regional effect, and were bilateral from unilateral injection.

Like many other workers, we found that sodium diatrizoate was the least toxic of the materials studied.

As regards other aspects of the chemical effects of contrast media, their toxicity is no doubt enhanced by certain conditions in the patient. Both hypotensive and hypertensive changes produced during the angiographic procedure, and in part due to the condition of the patient, may result in complications. There is general agreement that the concentration of the contrast medium in the blood is significant in reducing complications (Fig. 1).

Mechanical factors Mechanical factors in the production of angiographic complications have become more and more apparent. Since the angiography is now done so that the site of injection and the needle is included on the film, interstitial or subintimal injections, or an injection into the tissues of the neck, may be seen on the exposed film. Similarly when the needle is inserted directly through or into an atheromatous plaque, the course taken by the needle may be observed on the film. In one case the needle was inserted directly into the region of an atheromatous plaque in the neck. Immediately after injection this patient complained of inability to see toward the side of the injection. Clinically she had a homonymous field defect, and funduscopically occlusion of one of the main branches of the ophthalmic artery was observed. This visual field defect persisted in the form of a quadrant defect and it was felt that the embolus represented a so-called 'hard' or cholesterol embolus in contrast to a 'soft' or fibrin embolus which may well dissolve in a period of time.

Another complication arising directly from needle puncture is thrombosis with propagation. There is no question that in cases of atheromatous carotid stenosis any manipulation in the region of involvement can cause clot formation and propagation of thrombus. Another form of embolization is that of cotton fiber embolus (Fig. 2). These emboli obviously result from improper handling and cleaning of needles during the procedure and for the most part should be avoidable.

The improper placement of a needle in the carotid in the neck can result in intramural injection of contrast material with subsequent occlusion of the



Fig 4 a) Subdural injection. Needle was withdrawn and pressure applied over puncture site
b) Repeat injection 3 days later

vessel (Fig 3). When this is observed on the initial film, it is wise to withdraw the needle immediately and observe the patient closely for any developing neurologic deficits. Infrequently subdural injection can result in vessel occlusion and severe neurologic deficit. However, in a majority of cases simple compression at the site of injection for a period of minutes may result in absorption of the contrast material and re-establishment of normal flow within the artery (Fig 4).

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There are other mechanical effects unrelated to actual injections but directly related to manipulation by the point of a needle. We are somewhat surprised that more cases of jugular carotid fistulae have not been reported



Fig 5 a) Intramural injection b) Aneurysmal dilatation 3 weeks after (a)

following carotid angiography, since the introduction of the needle into both the jugular vein and the carotid artery is not an uncommon occurrence. We have had instances of Horner's syndrome following carotid angiography. This may either be due to the injection of local anesthetic into the sympathetic chain or it may result from direct trauma to the sympathetic chain from needle manipulation. Pneumothorax has occurred in both carotid and subclavian angiography. Local hematoma at the site of needle injection is a fairly common occurrence in both carotid and brachial angiography. Hematoma of surgical significance, however, is in our experience unusual. We have had one example of a patient who required tracheotomy following carotid angiography because of hematoma with tracheal compression and deviation and one example of massive cervical hematoma with dissection into the mediastinum resulting in death. We have examples of transient loss of radial pulse following retrograde brachial angiography, and have been told of one instance of occlusion of a brachial artery due to hemorrhage with subsequent infection, gangrene, and loss of the extremity.

Complications in patients with spontaneous subarachnoid hemorrhage from ruptured intracranial aneurysms are difficult to interpret. One is frequently asked whether the pressure of injection might not result in the re-

rupture of an aneurysm and subsequent bleeding. We have had only one example of contrast material extruding from a ruptured intracranial aneurysm during the angiographic procedure. This patient was almost moribund at the time of examination; the angiogram was obtained because of the possibility of a subdural or epidural hematoma.

Infection. Infectious causes for angiographic complications, in our experience have not occurred. We feel very fortunate in this regard and like to feel that it is due to good surgical techniques both in the preparation and handling of instruments used and in the rather meticulous surgical scrubbing prior to angiography. In our area we have seen one example of osteomyelitis of the cervical spine occurring after carotid angiography. We have also seen four cases of cervical abscess following carotid angiography, all of which resulted in the loss of the entire carotid complex on the side of infection.

Disease process and condition of patient

This category is perhaps the most difficult of all to interpret with respect to complications. There is agreement that the poor condition of the patient may be a contraindication to the employment of angiography. However, the disease process in a given case may help cause complications not ordinarily expected in a patient in relatively good condition. In two previous communications (LATIMER et coll 1952; LINDNER et coll 1962) we have stated that in the presence of angiographically demonstrable atheromatous changes in the vessels the complication rate for angiography is relatively higher. In an attempt to verify this stand we have reviewed several series of cases specifically with this in mind. One series of cases was selected only because both the carotid and vertebral arteries were examined on both sides (GURDJIAN et coll 1963). In 300 consecutive cases complications were noted in 14 instances. Hemiparesis occurred following carotid angiography in 2 cases. Both of these cases had contralateral carotid stenosis of 50% or greater noted on the angiogram. Another case of hemiparesis occurred following brachial angiography and in this instance generalized atheromatous changes were noted in both carotid and vertebral systems. Death occurred in one case in this series and angiography revealed complete middle cerebral occlusion. The patient at autopsy was found to have severe rheumatic heart disease and pulmonary edema. Cardiac fibrillation occurred following carotid angiography in one case examined for a possible subdural hematoma. Cervical hematoma of significant size was noted in another case in which carotid and vertebral atheromatous changes were seen on the films. Significant hypotension occurred in six instances.



Fig. 5. a) Intramural injection. b) Aneurysmal dilatation 3 weeks after (a)

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studies In this series any death occurring within twenty four hours of an angiographic study was considered a possible angiographic complication

On review of the total cases 8 of the 13 were felt to be directly related to angiography Of these eight patients cerebrovascular disease was a major diagnosis in five Of the remaining three cases one had a cardiac arrest during induction of anesthesia one was found to have a primary tumor of the pulmonary artery and the third had generalized carcinomatosis with cerebral metastases

In this same series of 1 000 cases the non fatal complications numbering eleven included 8 cases with major diagnosis of cerebrovascular disease In the remaining three one patient had an episode of coronary spasm when all of the contrast material at the time of carotid angiography went retrograde a second case developed heart block during operative exposure of the brachial arteries and a third case experienced radicular pain following percutaneous left cervical vertebral angiography

SUMMARY

Complications from cerebral angiography result from a number of interrelated mechanisms these include the chemical effects of contrast media the mechanical factors involved in manipulation of the vessels and needle insertion infectious causes and the existence of various disease processes in the patients which in one way or another contribute to some or all of the mechanisms mentioned above The most significant disease process in the production of most of the serious complications of angiography is the presence of cerebrovascular disease in particular atheromatous changes in the major vessels

ZUSAMMENFASSUNG

Komplikationen bei cerebraler Angiographie kommen durch eine Anzahl untereinander verwandter Mechanismen zustande Diese umfassen die chemischen Wirkungen von Kontrastmittel die mechanischen Faktoren wie Manipulation mit den Gefässen und Einfuhrung der Injektionsnadel Ursachen von Infektionen und Vorhandensein verschiedener krankhafter Zustände der Patienten die auf die eine oder andere Art be einigen oder allen oben genannten Mechanismen mitwirken Die wichtigste Erkrankung im Zusammenhang mit den ernststen Komplikationen bei der Angiographie ist das Vorhandensein einer cerebrovaskulären Erkrankung insbesondere atheromatose Veränderungen in den grosseren Gefässen

RÉSUMÉ

Les complications de l'angiographie cérébrale résultent de plusieurs mécanismes interdépendants ils comprennent les effets chimiques des moyens de contraste les facteurs mécaniques liés à la manipulation des vaisseaux et à l'introduction de l'aiguille les causes infectieuses et l'existence chez les malades de divers processus morbides qui d'une façon

The angiographic findings in the 1 000 patients with cerebrovascular disease were as follows

Internal carotid stenosis	204
Bilateral 97	
Internal carotid occlusion	91
Bilateral 10	
Anterior cerebral artery occlusion	118
Middle cerebral artery occlusion	38
Vertebral basilar occlusion	107
Aneurysm/arteriovenous malformation	37
Intracerebral hematoma (surgical)	23
Subdural hematoma	12
Brain tumor	40

In all of the patients, evidence of moderate to severe atheromatous changes was noted at angiography. All of the fourteen cases in this series having complications were in the sixth decade or later in life and all the cases having complications had a final diagnosis of some form of cerebrovascular disease. Five complications occurred associated with the use of general anesthesia, the remainder occurred during the use of local anesthesia.

Considering the disease categories in a series of 300 cases it was noted that 67 had demonstrable vascular disease in the carotid system, 26 in the vertebral basilar system, 81 were considered 'small artery disease', and 22 were being examined for possible aneurysm or arteriovenous malformation. The remaining 104 patients were brain tumor suspects or patients with vague non focal complaints, including convulsive disorders.

The overall morbidity of 4.1% and mortality of 0.3% may be compared with a second series of complications occurring in a group of 1 000 consecutive patients studied for cerebrovascular disease (LINDNER *et al.* 1962).

The angiographic complications in this series are summarized below.

Deaths	
Occlusive cerebrovascular disease	0
Aneurysm/arteriovenous malformation	2
Tumor of pulmonary artery	1
Metastatic carcinoma to brain	1
Hemorrhage or hemorrhagic infarction	2
Cardiac arrest	1
Transient weakness	4
Persisting weakness	2
Embolus	1
Coronary spasm	1
Radiculitis	1
Heart block	1
Convulsion	1

In this series an overall morbidity and mortality of 2.1% was counted with almost half of this figure representing possible mortalities from the

APPLICATIONS OF ANGIOGRAPHY DURING INTRACRANIAL OPERATION

by

JOHN W. LOOP and ELDON L. FOLTZ

Cerebral angiography is usually a pre operative examination but post operative angiography also provides valuable information about the brain circulation and the condition of brain vessels (MOUNT & TAVERAS 1956 NORLIN 1949). There are no valid studies to indicate the best time for angiography after operation. It has been the usual practice to delay it several weeks or months but we suggest that it might be more useful if the information gained by such an examination were available to the neurosurgeon immediately at operation. This would allow revision or extension of the operation as indicated by the angiogram. Particularly dangerous or complicated vascular lesions could thus be assessed several times during operation so that operative accomplishment could be immediately balanced against the risk of additional surgical treatment.

We have carried out cerebral angiography during operation for a variety of intracranial vascular diseases on ten patients to date. There have been no special technical problems. We have found it convenient to prepare and drape the neck separately from the cranium but to delay carotid puncture until immediately before contrast injection. If several injections have been planned the carotid cannula is continuously irrigated. Using a specially mounted manu-

ou d'une autre s'associent à certains ou à tous les mécanismes mentionnés ci-dessus. Le processus morbide le plus important dans l'apparition de la plupart des complications graves de l'angiographie est la présence d'une affection cérébro-vasculaire, en particulier les lésions athéromateuses des gros vaisseaux.

REFERENCES

- BROMAN T and OLSSON O: Tolerance of cerebrovascular vessels to contrast media. *Acta radiol* 30 (1948) 326
- LATIMER F R, WEBSTER J E and GURDJIAN F S: Complications of cerebral angiography. *Grace Hosp Bull* July (1952) 1—4
- LINDNER D W, HARDY W G, THOMAS L M and GURDJIAN F S: Angiographic complications in patients with cerebrovascular disease. *J Neurosurg* 14 (1962) 179
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- GURDJIAN F S, HARDY W G, LINDNER D W and THOMAS L M: Four vessel angiography. Experience with three hundred consecutive cases. *Clin Neurosurg* 10 (1963) 251

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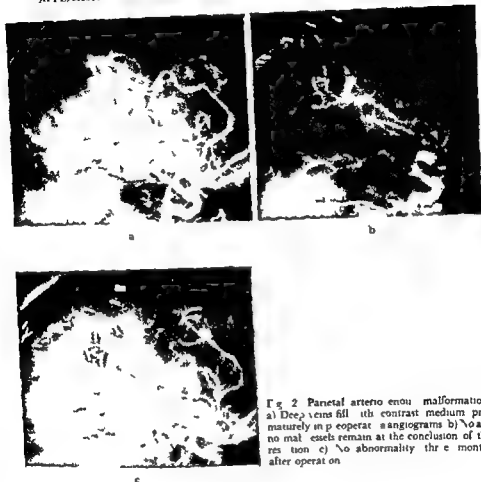


Fig 2 Parietal arteriovenous malformation
a) Deep veins fill with contrast medium prematurely in preoperative angiograms b) No abnormalities remain at the conclusion of the resection c) No abnormality three months after operation

Case 3 A 45 year old man had a twenty year history of increasing disability with left sided weakness and sensory loss. A very large right parietal arteriovenous malformation was demonstrated on preoperative angiograms. It could not be completely excised surgically although an attempt was made. Most of the large feeding arteries were interrupted. He improved temporarily but then developed right sided pulsating exophthalmos at which time angiograms showed striking enlargement of arteries which had formerly been small. He has since been treated by artificial embolization with opaque acrylic spheres introduced into the internal carotid artery as reported by LAURENCE et al (1962) (Fig 3). It is desirable for obvious reasons that the number of artificial emboli be limited to those which are useful in occluding pathological blood vessels. A composite illustration in this case shows the vascularity after two, after four and after eight acrylic spheres have been introduced into the circulation. Fig 4. The patient's proptosis is now gone. His hemiparesis persists but he states he is able to think more clearly and that he is more stable emotionally than preoperatively.



Fig 1 Internal carotid artery aneurysm. An angiogram during attempted ligation the neck of the aneurysm is not occluded.

ally operated cassette changer (DIE I, G. Schonander AB) under a sterile drape, we have been able to produce a series of three films. We have also occasionally used Polaroid film for quick viewing.

The following cases are presented to illustrate situations in which cerebral angiography during operation has been especially helpful to the neurosurgeon.

Case reports

Case 1 A 60 year old woman had sudden headache, right hemiplegia and aphasia. Intraoperative angiography revealed a large aneurysm of the left internal carotid artery with several daughter protrusions. It had a narrow neck on the angiogram and it was thought that this neck could be closed with a clip. However, at operation the neck of the aneurysm was thicker and stiffer than anticipated. The surgeon tightened a silk ligature about it as much as he dared. An angiogram was then made during operation and this showed the aneurysm filling as before (Fig 1). Having this information the surgeon immediately exercised his option to tie off the carotid artery in the neck. The patient has had no further bleeding in a year since the initial episode and neurologic deficits were only transient.

Case 2 A 21 year old man suddenly developed left hemiparesis. His right carotid angiogram showed arteriovenous shunting from anterior, middle and posterior cerebral arteries to the deep venous system (Fig 2a). His age, good general condition and the location of his lesion were indications for surgical excision (POOLE 1962). It is desirable to do the most conservative operation which removes the lesion completely. The angiogram assured the surgeon that he had left no malformed arteries deep to his resection (Fig 2b). No early venous filling was seen. He therefore terminated the operation. Three months later an angiogram showed an identical appearance (Fig 2c). The patient has had no clinical evidence of recurrence in a year.



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It is concluded that cerebral angiography during operation is useful in situations such as these. The time required to carry out the injection and expose the films does not add significantly to the length of the operation. No special complications have been encountered from angiography under these circumstances. On the other hand, in cases like those presented above, the neurosurgeon has been furnished with information at a time when it was most useful in order to carry out a more precise and definitive operative procedure.

SUMMARY

Three examples are presented in which cerebral angiography during operation provided information which was immediately helpful to the surgeon conducting the operation.

ZUSAMMENFASSUNG

Es werden drei Beispiele gezeigt, bei denen das Ergebnis einer cerebralen Angiographie, die während der Operation durchgeführt worden war, von unmittelbarem Wert für den operierenden Chirurgen war.

RÉSUMÉ

Présentation de trois cas où l'angiographie cérébrale peropératoire a fourni des renseignements immédiatement utiles au chirurgien qui opérait.

REFERENCES

- LUTSENHOP A. J., GIBBS M. and VELASQUEZ A. C. Cerebrovascular response to emboli. *Arch Neurol Psychiat* 7 (1962) 264.
- MOUNT L. A. and TAVERAS J. N. Cerebral angiographic studies following surgical treatment of intracranial aneurysms. *Acta radiol* 46 (1956) 333.
- NORLIN G. Arteriovenous aneurysms of the brain. Report of ten cases of total removal of the lesion. *J Neurosurg* 6 (1949) 475.
- POOLE J. L. Treatment of arteriovenous malformations of the cerebral hemispheres. *J Neurosurg* 19 (1962) 136.



Fig 3 Arteriovenous malformation a) The vessels have enlarged following an earlier operation b) Acrylic spheroids occlude the major tributary arteries



Fig 4 Arteriovenous malformation Angiograms obtained during operation after two (left view) four (centre) and eight (right view) spheroids introduced into the internal carotid artery have embolized the lesion

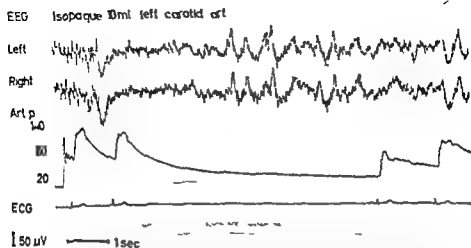


Fig 1 Carotid angiography with asystole for 6 sec and distinct fall of the blood pressure with bilateral electroencephalographic changes. The upper two curves show electrical brain activity in left and right frontal-parietal regions. The third curve represents the intra-arterial pressure in the common carotid artery. The lower curve is the electrocardiographic record.

In order to clarify both the circulatory effect and the brain wave changes in humans during cerebral angiography with different contrast media we have carried out the following investigations:

Material and method

In the past 2 1/2 years polygraphic recordings have been taken during cerebral angiography in 300 patients. In all 636 injections of contrast medium have been given in the carotid and vertebral arteries with the electroencephalogram, electrocardiogram and the intra-arterial blood pressure recorded simultaneously. Injections of 8 to 20 ml contrast medium were given percutaneously into the common carotid artery, into the vertebral artery or through a polyethylene catheter no. 160 inserted into the same arteries. Local anesthesia was used in most of the patients but general anesthesia was applied in special cases.

The comparison was as a rule made between two different contrast media and saline as a control in each patient. The sequence of injecting the two contrast solutions and a similar amount of saline was randomized to exclude the effect on the order of injections.

Out of this material 19 patients were excluded either because of only one

POLYGRAPHIC RECORDINGS DURING CEREBRAL ANGIOGRAPHY

by

ARNE LUNDEVOLD and ARNE FÅGESET

Cerebral angiography is not yet considered a completely innocent procedure and some complications both transient and permanent may occur. The contrast agents have therefore been tested in several ways. The effects on the EEG in both humans and laboratory animals were investigated by FOLTZ in 1952 and by THOMAS et coll. in 1954, who found either an immediate generalized flattening of electrical activity or homolateral production of new activity. INGVAR & SODERBERG (1957) examined both the EEG and the cerebral vaso motor tone.

GREITZ & WEISS (1959) investigated the influence of Hypaque and Mionon on the EEG and ECG in cerebral angiography. They found like INGVAR & SODERBERG, that the EEG was unchanged, but in the ECG rhythmical disturbances were recorded in some patients.

The circulatory effect in general of intracarotid injections of one particular or other contrast materials has been described by several authors (BROMAN & OLSSON 1948, HAUGE 1951, GREITZ 1956, LINDGREN & TORNELL 1958, FISHER & ECKSTEIN 1961 & 1962). On the other hand BULL & MURRAY LESLIE (1962) do not agree with all the findings in their animal studies and stress that one cannot place too much reliance on animal experiments alone.

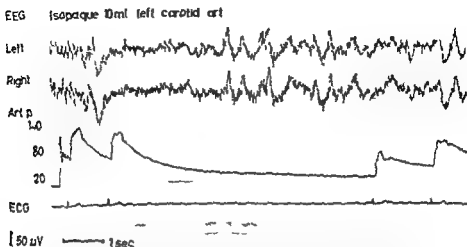


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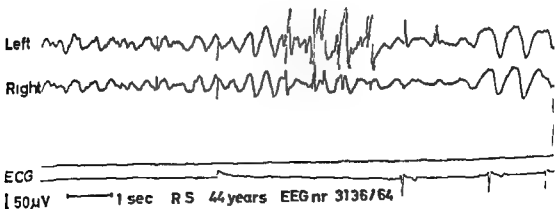
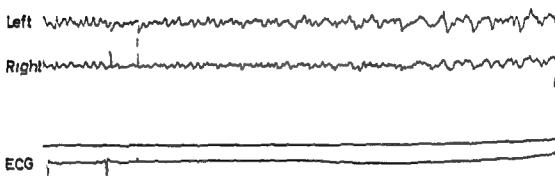
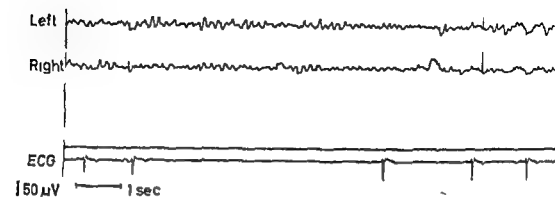
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b

Fig 2 a) Vertebral angiography. The first injection of Isopaque 45 resulted in a 6 sec asystole
 b) Second injection in same patient resulted in a 19 sec asystole

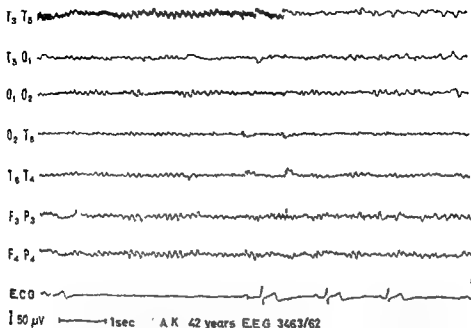


Fig 3 a Carotid angiography (Hypaque 10 ml) with asystole for 4 sec and a simultaneous focal electroencephalographic changes with accompanying transient paresis of the extremities contralaterally. The upper three curves and the sixth curve show the slow waves over the left hemisphere. The other curves show normal activity over the right hemisphere.

injection a defective filling or for other technical reasons. The distribution of the remaining 281 patients was as follows:

	Total	Female	Male	Right side	Left side
Carotid angiography	262	95	167	127	135
Cerebral angiography	19	8	11	8	11
Total	281	103	178	135	146

Results

The patients reacted generally very little to the injections as the EEG, ECG and blood pressure remained unchanged.

In some tense patients however we noted an increase in the blood pressure as well as an increased cardiac rate with a decrease of voltage in the EEG as often seen in arousal or in frightened patients. All these responses or lack of responses were considered normal.

A typical positive response consists of marked slowing of the heart rate

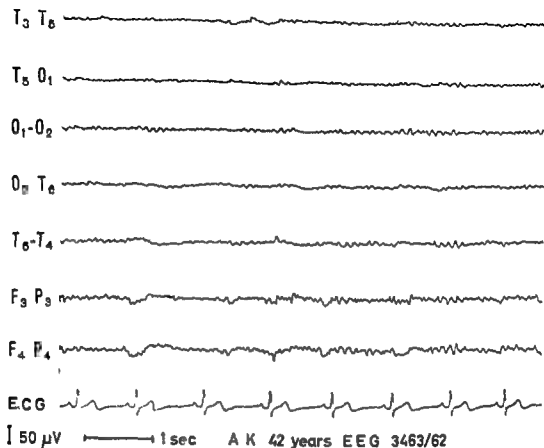


Fig 3b The following injection of saline in same patient gave no abnormal responses

and/or a brief period of asystole, starting 3 to 5 sec after the beginning of the injection (Fig 1)

In our tables only periods of asystole lasting more than 1 1/2 sec are shown because measuring slowing of the heart rate depends too much on the time over which the heart beats are counted. Associated with asystole or slowing of the heart rate was a fall in the blood pressure, the magnitude of which correlates with the severity and duration of the drop in heart rate (Fig 1)

These responses do not appear to be caused by physical or chemical stimulation of the carotid sinus. The effects on blood pressure and cardiac rate were present even when the carotid sinus was anesthetized, the catheter placed far beyond the sinus, or, most convincingly, when the contrast medium was injected into the vertebral artery, as shown in Fig 2 a

Even with a 15 minute period between the injections, the second injection gave, as a rule, more abnormal responses (Fig 2 b)

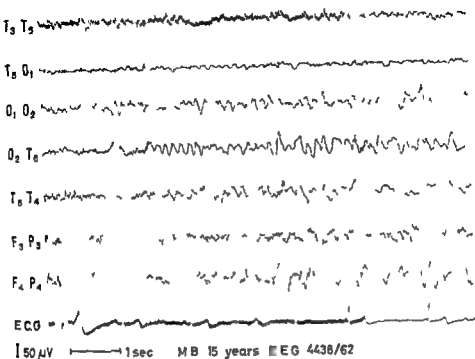


Fig 4 a Carotid angiography (Isopaque 10 ml) with focal electroencephalographic changes lasting 5 min with accompanying transient paralysis of the extremities contralaterally. Abbreviation the same as in fig 3 a

The long lasting asystole caused drastic changes in the EEG as well

These abnormalities in the polygraphic recordings did not occur with injections of saline with compression of the carotid artery or palpation of the carotid sinus

We therefore believe that the slowing of the heart rate and the lowering of the blood pressure are caused by direct action of the contrast medium on the brain probably the hypothalamus the brain stem or other brain structures being supplied by the posterior cerebral artery as a much higher incidence of fall in blood pressure and slowing of the cardiac rate was noted when this artery was filled with contrast medium both in cerebral and vertebral angiography than when it was not seen (cf GREITZ). Other supporting evidence for this theory was found in the electroencephalographic findings

Fig 3a shows that in addition to the slowing (or asystole) of the heart rate a slowing of the brain waves also was recorded on the same side as

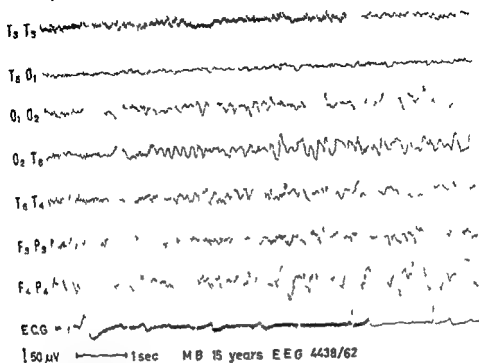


Fig 4a Cerebral angiography (Isopaque 10 ml) with focal electroencephalographic changes lasting 5 min with accompanying transient paralysis of the extremities contralaterally. Abbreviation the same as in fig 3a

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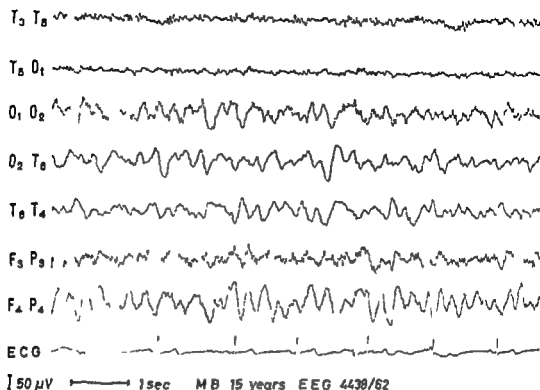


Fig. 1b. Curves obtained 10 sec later.

that on which the injection was performed. The following injection of the same amount of saline gave no abnormal responses (Fig. 3b). If the above normalities in the electroencephalograms lasted for several minutes they were followed by a contralateral hemiparesis and positive Babinski signs (Fig. 4, a and b). These clinical signs were transient, but we had two other patients with probably permanent hemiparesis.

In the first 106 patients, 10 ml Hypaque 45%, 10 ml Isopaque 45%, and 10 ml saline were injected in different sequences (see below).

	Total	Female	Male	Right side	Left side
Percutaneously	37	13	24	23	14
Catheter method	69	22	47	31	38
Total	106	35	71	54	52

There were no significant changes in the brain waves, heart rate or blood pressure responses (Table 1).

We observed, however, that there was a tendency for the contrast medium being injected through a polyethylene catheter to give more abnormal responses

Table 1

ECG and EEG abnormalities in 106 patients during 231 injections of contrast medium at cerebral angiography

	Asystole > 1 1/2 sec	Abnormal EEG	Total
Hypaque 45	17	18	35
Isopaque 45	16	20	36
Total	33	38	71

than the percutaneous injections. Despite this the catheter method was used for the most part in the remaining group of patients primarily to prevent a dissecting aneurysm which sometimes happens with the percutaneous method. Another reason is that the patients are much easier to handle during the roentgen procedure with a catheter rather than a needle in the carotid artery.

In order to diminish or avoid these deleterious effects of the contrast medium of the brain a new more physiologic solution was composed in cooperation with the Norwegian firm Nyegaard & Co. A/S and called Isopaque B. It is based on the sodium salt of metrizoic acid and contains Ca and Mg in the same relative concentration as the human plasma.

Animal experiments using rabbits and the same method as with the patients were first carried out. In 26 rabbits of an average weight of 3 kg 2 ml Isopaque 45% or 2 ml Isopaque B 45% were injected in the carotid artery every third minute 6 injections in all or until the rabbit expired. The injections were done under Urethan narcosis. Table 2 shows the grade of complications.

After these and other animal experiments had shown that the new medium Isopaque B was superior to the old one it was introduced into human angiography.

In order to compare the two contrast media in human cerebral angiography both were injected every other time in alternate order interrupted by injec-

Table 2

ECG and EEG abnormalities in 26 rabbits during 173 injections of contrast medium in the carotid artery

	ECG changes		EEG changes		Total
	Markedly abnormal	Moderately abnormal	Markedly abnormal	Moderately abnormal	
Hypaque 45	4	3	6	1	14
Isopaque B 45	0	3	2	4	7

Table 3

ECG and EEG abnormalities in 175 patients during 405 injections of contrast medium at cerebral angiography

	Asystole $> 1\frac{1}{2}$ sec	Abnormal EEG
Isopaque 45 % only	19	16
Isopaque II 45 % only	3	7
Isopaque and Isopaque II	13	3
Total	35	26

tions of the same amount of saline. In 175 patients, injections of Isopaque 45 % and Isopaque B 45 % were given as follows

	Right side	Left side	Total
Female	29	39	68
Male	52	55	107
Total	81	94	175

The results are shown in Table 3

The two sexes were compared as well as the two injection sides, but no significant changes were found. Compression of the common carotid artery on one side gave no more positive responses.

The age of the patients, the diagnosis, the premedication and the type of

Table 4

	Cerebral angiography Total	Complications with Hypaque 45 % and Isopaque 45 %	Complications with Isopaque II 45 %
Average age	37 $\frac{1}{2}$ years	45 years	50 $\frac{1}{2}$ years
Tumor intracranialis	31	46 %	56 %
Vascular lesions	26 %	14 %	11
Epilepsy	22 %	20	
Miscellaneous	21	20 %	33 %
Narcosis	24 %	17	6 %
Premedication			
Atropine	17	15	11 %
Morphine scopolamine	11 %	10 %	
Amytal			
Acid Isoamyläthylbarbituric	72 %	75	89

¹ Only one patient with asystole

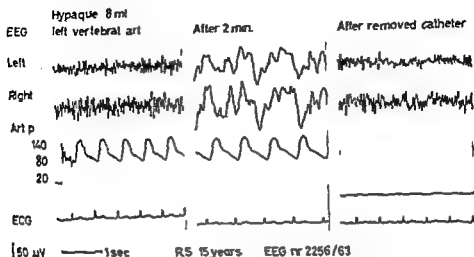


Fig 5 Left curve shows that injection of contrast medium through a catheter in the vertebral artery does not produce changes in electroencephalogram (upper two curves) intra arterial blood pressure or electrocardiogram. Left curve shows marked pathological activity in electroencephalogram recorded after catheter probably blocked obstructed vertebral artery for 2 min. Right curve shows normal electroencephalogram taken immediately after withdrawal of catheter.

anesthesia may influence the results. Table 4 gives the number of patients in each of these groups classified after the polygraphic results.

This table indicates also that Isopaque B is a better contrast medium than the ordinary type. The average age is higher in the patients with complications and again highest in the group with complications after Isopaque B.

In our material the anesthesia does not seem to have an unfavourable effect. The same is the case with patients who have vascular lesions. On the other hand we found more patients with tumors in the group with complications and especially with complication following injections of Isopaque B.

We believed that the premedication was very important because other investigators (FISHER & PIRRET 1962) found in animal experiments that the vascular changes could be abolished by sectioning the vagus nerve. In man, protection from the circulatory disturbances might be obtained with atropine blocking of the vagal responses (AMUNDSEN & AKEVES).

In our investigations atropine in general had a tendency to improve the results but it did not prevent all the complications especially not the EEG changes. 6 of the 7 patients with complications had abnormal EEG recordings and only one patient had asystole for 1 1/2 seconds. It is possible that even better results could be obtained with a more adequate dose of atropine.

Table 3

LCC and FFC abnormalities in 175 patients during 405 injections of contrast medium at cerebral angiography

	Asystole > 1 1/2 sec	Abnormal FFG
Isopaque 45° only	19	16
Isopaque B 45° only	3	7
Isopaque and Isopaque B	13	3
Total	35	26

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Female	29	39	68
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The results are shown in Table 3.

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Premedication			
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Morphine scopolamine	11°	10	
Amical			
Acid Isoamylmethylbarbituric	72°	75	89°

¹ Only one patient with asystole.

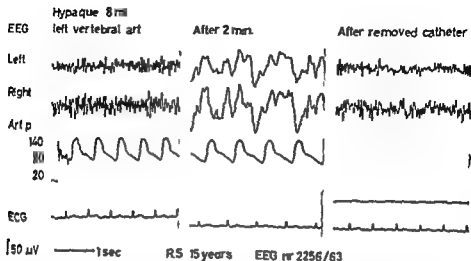


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We believed that the premedication was very important because other investigators (FISHER & PIRRET 1962) found in animal experiments that the vascular changes could be abolished by sectioning the vagus nerve. In man protection from the circulatory disturbances might be obtained with atropine blocking of the vagal responses (AMUNDSEN & ARNES).

In our investigations atropine in general had a tendency to improve the results but it did not prevent all the complications especially not the EEG changes. 6 of the 7 patients with complications had abnormal EEG recordings and only one patient had asystole for 1 1/2 seconds. It is possible that even better results could be obtained with a more adequate dose of atropine.

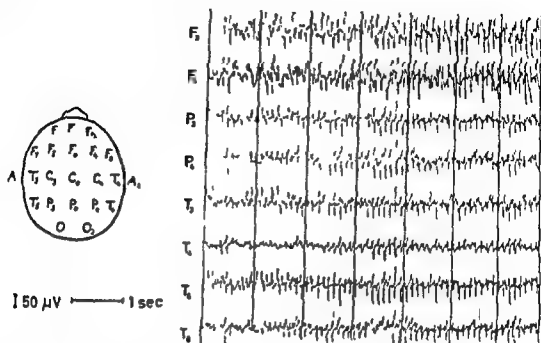


Fig 6 Seizure activity during general anesthesia with curare before angiography. No clinical seizure signs visible after angiography when curare effect had disappeared

Even if the new contrast medium, Isopaque B, seems to be better than the previous one, it is not at all harmless to the brain. Our radiological department has taken this into consideration and uses routinely polygraphic registration during angiography in all patients suspected of developing trouble or complications at the angiography. Especially in view of the fact that the second or third injection seems to be more dangerous than the first, we believe that some patients have been spared transient or longer lasting brain damage, due to the warnings given by the polygraphic recordings.

Not all the complications during angiography are caused by the contrast medium, however. Fig 5 demonstrates that the obstruction produced by the catheter in the vertebral artery may cause very marked changes in the EEG, followed by unconsciousness. In this case it was probably caused by a vessel anomaly as most, or all, of the blood supply to the basilar artery came from the obstructed vertebral artery.

With patients having general anesthesia during angiography we have often used the polygraph throughout the whole procedure. Fig 6 shows an electroencephalographically recorded seizure without clinical signs, as the patient was in general anesthesia, and curarized. The EEG seizure started during the general anesthesia before the angiography, but first after the effect of the curare had disappeared were the clinical signs observed.

Addendum in proofs

Since these investigations were completed some 200 more patients have been examined in the same way but the amount of contrast medium has been reduced from 10 to 7 ml. The number of injections have also been minimized as much as possible.

Presumably because of the reduced amount of a less toxic contrast medium the premedication with atropine and by reducing the intracranial pressure with Mannitol or Urea in patients with expanding lesions the changes in the polygraphic recordings have been reduced considerably.

Among the first 100 patients about one third had changes in the ECG and about the same number had EEG disturbances.

Because of the alteration in the roentgenological procedure mentioned above only 10% of the last 100 patients had such changes and these were minimal compared with those among the first 100 patients.

SUMMARY

Polygraphic recordings during cerebral angiography in 300 patients have shown marked changes in the electroencephalogram, electrocardiogram and intra arterial blood pressure. Electrocardiographic changes were found in 68 patients. Focal slow waves in the electroencephalograms often combined with slight and more rarely with marked transient signs of hemiparesis were found in 64 patients when Hypaque 45% and Isopaque 40% was used as contrast medium. The obstruction effect of the catheter could also produce slow waves in the EEG. In a later similar study of 200 patients the polygraphic changes were reduced to about 10% and were not as marked. The cause was probably the reduction in the amount of contrast media from 10 to 7 ml, the premedication with atropine and the reduction of the intracranial pressure in patients with expanding lesions.

ZUSAMMENFASSUNG

An 300 Fällen wurden während der cerebralen Angiographie polygraphische Aufzeichnungen vorgenommen. Die erhebliche Veränderungen im Electro-encephalogramm, im Elektrokardiogramm und im intra arteriellen Blutdruck zeigten elektrokardiographische Veränderungen wurden 68 mal gefunden. Bei 64 Patienten zeigten sich nach Hypaque 45% oder Isopaque 45% örtliche verzögerte Wellen im EEG, die öfter mit leichten Zeichen selten mit schwereren aber vorübergehenden Zeichen einer Hemiparese verknüpft waren. Der Obstruktionseffekt des Katheters konnte ebenfalls verlangsamte EEG Wellen hervorrufen. Eine neue Serie von 200 Patienten zeigte die Nebenerscheinungen nur in 10% der Fälle und die Nebenerscheinungen waren milderer Natur. Die Ursache war wahrscheinlich, dass der Betrag des Kontrastmittels von 10 ml auf 7 ml reduziert wurde, dass wir mit Atropin prämedizierten und den Hirndruck bei raumbegrenzenden Prozesse vorbeugend herabbrachten.

RÉSUMÉ

Les enregistrements polygraphiques au cours de l'angiographie cérébrale chez 300 sujets ont montré des altérations marquées de l'électro-encephalogramme, de l'électrocardiogramme et de la pression intra artérielle. On a trouvé des modifications électrocardiographiques chez 68 malades. On a trouvé sur les électro-encéphalogrammes des ondes lentes focales souvent

associées à des signes légers et plus rarement marqués d'hémiplégie transitoire quand on utilise comme moyen de contraste l'Hypaque 45 % et l'Isopaque 45 %. L'obstruction par le cathéter peut aussi donner des ondes lentes sur l'EFG. Dans une étude similaire ultérieure sur 200 sujets les altérations polygraphiques étaient réduites à environ 10 % et étaient moins marquées. La cause en est probablement la réduction de la quantité de moyen de contraste de 10 à 7 ml la prémédication par l'atropine et la réduction de la pression intracrânienne chez les malades atteints de tumeurs.

REFERENCES

- AMUNDSEN P and AASNES F. Personal communication.
- BROMAN T and OLSSON O. Tolerance of cerebral blood vessels to contrast medium of diodrast group: experimental study of effect on blood brain barrier. *Acta radiol* 30 (1948) 376.
- BULL J W D and MURRAY LESTER R M. Experience with metrizoate sodium (Isopaque) in cerebral angiography. A report on 33 cases. *Gammaoterapi* 1 (1962) 24.
- FISCHER H W and FENSTER J W. Comparison of cerebral angiographic contrast media by their circulatory effects. *Amer J Roentgenol* 86 (1961) 166.
- — and PIRRET G. Comparison of the cardiovascular effects of contrast media in cerebral angiography. *in* *man* *J Neurosurg* 19 (1962) 943.
- FOLTZ E L. The EEG effects of intracarotid diodrast. *Electroenceph clin Neurophysiol* 1 (1952) 113.
- GRFITZ T A. Radiological study of brain circulation by rapid serial angiography of carotid artery. *Acta radiol Suppl* 140 (1956).
- — and WEISS S. EFG and ECG in cerebral angiography with sodium diatrizoate (Hypaque) and methylglucamine diatrizoate (Miolon). *Acta radiol* 52 (1959) 145.
- HAUG T. Catheter vertebral angiography. *Acta radiol Suppl* 109 (1954).
- INGVAR D H and SODERBERG U. Cerebral vasomotor tone and EEG during injection of umbradil: experimental study with new method. *Acta radiol* 47 (1957) 185.
- LINDGREN P and TORNELI G. Blood circulation during and after peripheral arteriography. *Acta radiol* 49 (1958) 425.
- — Blood pressure and heart rate responses in carotid angiography with sodium acetrizate (triurof) experimental study in cats. *Acta radiol* 50 (1958) 160.
- LUNDKVOLD A, SARAASSTAD T and BORCHSEN A. Cerebral angiography with simultaneous electroencephalographic, electrocardiographic and electromyographic recordings and measurement of intra arterial pressure. *Nord Med* 70 (1963) 853.
- THOMAS L H, FOLTZ E L and WARD JR A A. The modification of cerebral electrical activity by intracarotid and topically applied diodrast. *Electroenceph clin Neurophysiol* 6 (1954) 643.

ANGIOGRAPHIC OBSERVATIONS FOLLOWING REMOVAL OF SUBDURAL HEMATOMAS

by

ROBERT L. McLAURIN and FREDRIC HELMER

The observations to be reported were made from serial angiographic studies on patients with subdural hematomas. This is part of a larger project designed to investigate the usefulness of angiography following removal of benign expanding intracranial lesions. Although pneumography has been used previously to follow the migration of intracranial structures there are limitations to the usefulness and value of that type of approach. The purposes of the present study were: 1) to delineate the normal anatomy as a base line against which comparison may be made in cases of suspected post operative recurrent hematomas and 2) to compare the appearance of the angiograms with clinical manifestations in order to understand neurologic changes occurring after hematoma removal.

The material studied included angiograms on 19 patients. Of these 18 had surgical evacuation of hematomas while 1 patient was followed through the period of complete spontaneous resolution of the hematoma. In each case 1 preoperative angiogram was performed and 1 to 4 post-operative studies carried out.

Return of the angiographic appearances to normal following evacuation of a subdural hematoma may be divided into two components: 1) return of the

associés à des signes légers et plus rarement marqués d'hémi-parésie transitoire quand on utilise comme moyen de contraste l'Hypaque 45 % et l'Isopaque 45 %. L'obstruction par le cathéter peut aussi donner des ondes lentes sur l'EEG. Dans une étude similaire ultérieure sur 200 sujets les altérations polygraphiques étaient réduites à environ 10 %, et étaient moins marquées. La cause en est probablement la réduction de la quantité de moyen de contraste de 10 à 7 ml la prémédication par l'atropine et la réduction de la pression intra-crânienne chez les malades atteints de tumeurs.

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- FISCHER H W and FICKSTEIN J W. Comparison of cerebral angiographic contrast media by their circulatory effects. *Amer J Roentgenol* 86 (1961) 166.
- — and PIRRET G. Comparison of the cardiovascular effects of contrast media in cerebral angiography. *in Ann J Neurosurg* 19 (1962) 913.
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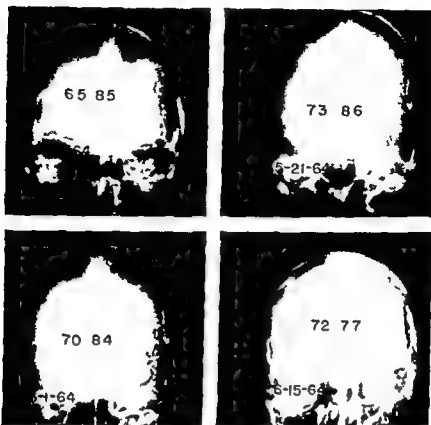


Fig 1 Case 1 Serial left carotid angiograms. Slow recovery of midline position of anterior cerebral artery in spite of almost complete absence of subdural accumulation post operatively. The numbers refer to measured distances of anterior cerebral artery from inner skull table. Dates of studies are also shown. Surgical drainage was performed on May 1, 1964.

pericallosal artery to the midline, indicating recovery from shift of the vital midline structures, and 2) disappearance of the surface accumulation of fluid. These aspects of arteriographic recovery may proceed independently, suggesting that shift of the midline vessels is not solely due to the surface hematoma. In general, clinical recovery seemed to be related more closely to migration of the pericallosal artery to the midline position, although exceptions to this occurred in patients who made striking clinical recovery despite continued displacement of the midline structures.

In 2 instances the pericallosal artery did not fill on the preoperative study and in 1 of these it was in midline position on the following day. In all patients in whom displacement was demonstrated preoperatively the minimum time required for return to midline was 8 days and in most instances 2 to 4 weeks were required. In 1 patient there was still a definite shift after 60 days and in this instance the clinical recovery progressed with reduction of the size of the



Fig 2 Case 2. Serial right carotid angiograms. Absence of subdural accumulation 5 days post-operatively and return of anterior cerebral artery to normal position at 2 weeks. Surgical drainage was done on Oct 31 1963.

surface hematoma and lateral migration of the sylvian point. There was no indication that age modified the speed of post-operative migration in any way.

Four patients have been selected to illustrate several of the points observed in this study.

Case reports

Case 1 C. R. a 67-year-old male was admitted to the hospital on April 25 1964 following an auto accident. On admission he was slightly confused but otherwise intact neurologically. During the next few days a right hemiparesis and dysphasia appeared but later these seemed to subside. Three weeks after admission the hemiparesis increased, the conscious level declined and a carotid angiography demonstrated a left parieto-temporal hematoma. The hematoma was immediately evacuated and thereafter he progressively and completely recovered. Serial post-operative angiographies were performed during the month following operation (Fig 1).

Six days following surgical intervention the sylvian point had returned to normal position and there was only a small residual accumulation despite continued marked displacement of the pericallosal artery. It was still significantly displaced after 17 days and at 1 month had just returned to within normal limits. The relatively slow return of the pericallosal artery in the presence of rapid return of the sylvian point is undoubtedly a reflection of the slow recession of edema of the cerebral substance.

Case 2 N. C. a 62-year-old male was hospitalized on Oct 17 1963 following a fall of unknown cause. On admission he was deeply comatose and had a left hemiplegia. During the next few days he demonstrated a marked recovery of function of the paretic extremities and 6 days after injury he was ambulatory. A diagnosis of probable cerebrovascular occlusion disease was made and 2 weeks after admission carotid angiography was performed disclosing the presence of a large right subdural accumulation. This was immediately evacuated and 2 post-operative angiographies were done (Fig 2). Clinical recovery continued slowly during the 2 weeks following operation.

Five days following surgical intervention there was no demonstrable surface accumulation although the pericallosal artery was still significantly displaced. Two weeks after surgery the vessel had returned to normal position. The sylvian point originally displaced inward by approximately 2 cm returned to normal within 5 days. Correlation of the clinical recovery

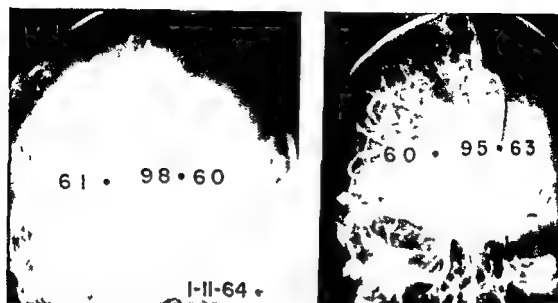


Fig 3 Case 3 Pre and post operative right carotid angiograms show only slight change of vascular displacement despite almost complete neurologic recovery. The figures at left indicate distance of sylvian point from inner skull table. Surgical drainage was done on Jan 11 1964.

with the angiographic findings is hazardous since it seems quite probable in view of pre-operative improvement that cerebral contusion was responsible for some of the deficit. Nevertheless it appears that contusion and/or swelling of the hemisphere accounts for most of the neurologic disturbance rather than the surface pressure.

Case 3 II O a 32 year old female a chronic alcoholic had fallen several times and complained of headache for about 7 days before admission to the hospital on Jan 11 1964. During the 2 days prior to admission she had gradually become less responsive. On admission she was comatose had dilatation of the right pupil and right hemiparesis. Carotid angiography demonstrated the presence of a large right subdural hematoma which was evacuated immediately. On the following day she was markedly improved and awake with no motor weakness. Five days after surgery a post operative angiography was carried out (Fig 3).

The significance of this case is the striking clinical recovery despite continued presence of marked shift of the pericallosal artery and inward displacement of the sylvian point. It would appear that surgical intervention relieved the critical degree of intracranial displacement only. It is possible that if she had never reached this critical displacement initially spontaneous recovery might have occurred. At all events the observations in this case emphasize the lack of predictable relationship between intracranial vascular displacement and clinical manifestations.

Case 4 H B a 60 year old male was admitted to the hospital on March 24 1964 because of confusion and disorientation associated with a history of chronic alcoholism. Examination revealed aphasia and pathologic reflexes on the right. Roentgenograms disclosed a pineal shift from left to right and carotid angiography showed a subdural hematoma. Since the patient was demonstrating no neurologic deterioration it was decided to postpone surgical intervention. During the ensuing 6 weeks 5 angiographies were done and there was gradual return of

vessels to the normal position with simultaneous disappearance of the subdural accumulation. Clinically he made a progressive and complete recovery. Following arteriographic recovery it was felt that the diagnosis should be verified and a simple trephine opening disclosed a membrane 3 mm in thickness consistent with that associated with a subdural hematoma.

It appears that this case represents another example of a spontaneously subsiding subdural hematoma. The operative findings indicate that the surface accumulation was not simply a subdural hydroma which is not accompanied by thick membrane formation. Similar cases have been previously reported by AMBROSETTO (1962) and by GANNON, COOK and BROWDER (1962). The frequency with which spontaneous resolution may occur is not known. It seems logical that it would occur in certain instances as we have clearly demonstrated that subdural accumulation and cerebral displacement is frequently present after surgical drainage of a subdural hematoma.

Discussion

Two previous studies comparable to this investigation should be mentioned. PARINSON & CHOCKINOV in 1960 observed the migration of silver clips placed on the cortex and dura. With chronic hematomas a 2 to 5 mm reduction of the arterial displacement occurred during the first 24 hours and during the next 10 days there was very little further reduction although the patients continued to improve. The authors concluded that the initial few millimeters is all that is needed to initiate recovery. Our observations would support this concept but we believe that subsequent recovery is due to continued midline migration which is largely the result of edema of the involved hemisphere.

In 1962 COOK, BROWDER and CARTER reported a series of encephalograms on 22 patients with post traumatic hematomas and 1 hydroma. In patients treated by craniotomy the shift lasted a shorter time and the authors mentioned persistence of subdural accumulation as a factor contributory to the midline shift.

Thus each of the above methods of study measures a separate factor in post operative recovery but neither measures both factors. The present authors believe that persistence of midline shift and gradual neurologic recovery are due to a combination of subdural reaccumulation and underlying cerebral swelling. Resolution of these components occurs at independent rates. Carotid angiography provides a method of demonstrating the two components simultaneously and therefore contributes to understanding the post operative recovery process.

BROWDER & RABINER (1951) have written extensively about the cerebral edema which exists beneath a subdural hematoma. They demonstrated focal subcortical swelling beneath hematomas in patients dying after intervals of at least 4 days following trauma while no swelling was noted in patients dying of subdural hematomas in less than 4 days. The swelling accounts for 1) rapid neurologic deterioration late after injury, 2) failure of prompt clinical recovery

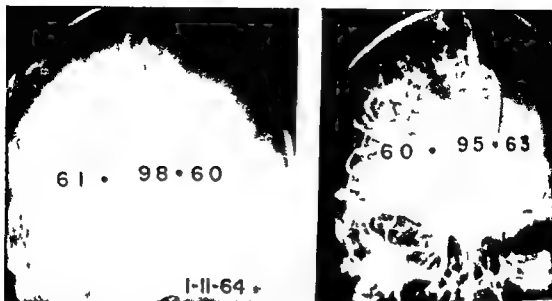


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ont l'hématome a été évacué l'artère péricalléuse revient à sa place normale en 2 à 4 semaines. La persistance d'un déplacement de cette artère après l'opération est due à l'association d'un oedème cérébral et d'un nouvel épanchement sous dural. Ces facteurs disparaissent avec des vitesses indépendantes. Ces mêmes facteurs, ainsi que l'insuffisance vasculaire due à la compression, contribuent au déficit neurologique et à sa disparition post-opératoire.

REFERENCES

- AMBROSETTO C. Post traumatic subdural hematoma. Arch Neurol 11 (1962) 287
- BROWDER E. J. and RABINER A. M. Regional swelling of the brain in subdural hematoma. Ann Surg 134 (1951) 369
- COOK A. W. BROWDER E. J. and CARTER W. B. Cerebral swelling and ventricular alterations following evacuation of intracranial extracerebral hematoma. J Neurosurg 19 (1962) 419
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- LINDENBERG R. Compression of brain arteries as pathogenetic factors for tissue necrosis and their areas of predilection. J Neuropath Exp Neurol 14 (1955) 223
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Three patients in the present series have shown some interference with filling of the pericallosal artery in the presence of a subdural hematoma with subsequent filling after hematoma evacuation. The possibility exists that these may have been chance occurrences but the authors are inclined to the belief that some vascular interference resulted from the shift of intracranial contents. LINDENBERG (1955) has reported extensive observations on vascular compression during intracranial displacement and noted that the anterior cerebral artery may be compressed by the gyrus rectus and that the pericallosal artery is compressed by herniation beneath the falx. Whatever the mechanism of compression its presence may contribute to the neurologic deficit and the critical degree of displacement which causes neurologic decompensation may be that which produces sufficient vascular insufficiency.

SUMMARY

Observations in angiographic studies on 19 patients with subdural hematomas are recorded. Fifteen underwent surgical evacuation while 1 demonstrated spontaneous hematoma disappearance. In patients whose hematomas were drained the pericallosal artery returned to normal position in 2 to 4 weeks in most instances. Continued post operative displacement is due to a combination of cerebral edema and subdural reaccumulation; these factors subside at independent rates. These same factors as well as vascular insufficiency from compression contribute to the neurologic deficit and its post operative subsidence.

ZUSAMMENFASSUNG

Angiographischen Studien bei 19 Patienten mit subduralen Hämatomen werden berichtet. Achzehn wurden operiert während sich in einem Fall das Hämatom spontan zurückbildete. Bei den Patienten bei denen die Hämatoome drainiert wurden kehrte die Art. pericallosa in den meisten Fällen in 2 bis 4 Wochen zur normalen Lage zurück. Fortdauernde postoperative Verlagerung ist auf eine Kombination von cerebralem Ödem und subduraler Wiederauffüllung zurückzuführen. Diese Faktoren bilden sich unabhängig voneinander mit verschiedener Geschwindigkeit zurück. Dieselben Faktoren wie auch vaskuläre Insuffizienz nach einer Kompression leisten zum neurologischen Ausfallsymptom und seiner postoperativen Rückbildung einen Beitrag.

RÉSUMÉ

Les auteurs présentent des remarques sur l'étude angiographique de 19 cas d'hématome sous-dural. L'évacuation chirurgicale de l'hématome a été faite dans dix-huit cas et la disparition spontanée de l'hématome s'est produite dans un cas. Chez la plupart des malades

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agent intended to act locally upon the vascular bed or parenchyma of the cord no matter how administered. These considerations led them to conclude that any hope for an effective therapy for the adverse effects of roentgen contrast agents would reside in prophylactic rather than remedial approaches.

In a series of titrations of the toxicity of various dilutions of fixed volume per kilogram doses of Na acetrizolate they were impressed by two observations (40-62): 1) That concentration was the major factor in the production of injury and 2) correlation between progressive concentration and injury was not parallel. Instead there was an abrupt demarcation between innocuous and toxic effects almost approaching an all or none relationship in some tests. This non linear relationship could be readily explained by the vasotoxic reaction as follows: even an instantaneous exposure of the vascular bed of the cord to a toxic concentration of contrast agent will initiate a series of circulatory events leading to irreversible tissue injury. The vasotoxic reaction of itself will increase the application time five to tenfold and the subsequent changes in vascular dynamics of the cord will further intensify the injury (42). It was observed also that the supine position during injection favored the production of spinal cord injury, an effect interpreted as the result of differential gravitational flow of the contrast agent with a resultant higher concentration reaching the vascular bed of the cord.

Holding that all contrast agents were capable of producing a necrotizing lesion if they reached the vascular bed of organs in the mainstream of their flow in concentrations approaching the high initial level required for angiography they initiated a series of studies exploring means of protecting recipient subjects (24-64). Information derived from these investigations would also provide insight into the mechanisms of tissue injury caused by these substances. Measuring therapeutic action against the rigorous test of a 2 ml/kg dose of 70% Na acetrizolate (Urokon) in their experimental model of retrograde abdominal aortography they observed dramatic protective effects after prophylactic administration of a group of vasodepressor drugs of varied loci of action. These included two local analgesics (Procaine HCl, Carbocaine HCl), a ganglionic blocking agent (trimethaphan camphor sulfonate [Arfonad]) and a smooth muscle relaxant (Papaverine). Considering that they were dealing with a concentration dependent toxic action they concluded that the protective effect of these vasodepressor drugs resulted from the diversion of the intra aortic injection mass to the periphery hence diluting it below toxic levels in the vascular bed of the spinal cord.

If these interpretations of the mechanism of protective action of vasodepressor agents against neurotoxic effects of contrast agents are correct it would be expected that a converse effect — a potentiation of the neurotoxicity of contrast

VASOPRESSOR POTENTIATION OF NEUROTOXICITY IN EXPERIMENTAL AORTOGRAPHY

Implications regarding pathogenesis of contrast medium injury

by

GEORGE MARGOLIS and THEODORE G. YERASIMIDES

In 1956 MARGOLIS and associates introduced a new experimental model (39, 60) for the study of the pathogenesis of the injury to the nervous system produced by contrast media used in diagnostic angiography. Employing retrograde abdominal aortography in the dog, and using the spinal cord as the site of study they described, for the first time in experimental animals, the full evolution of the contrast medium lesion. From these observations it was manifest that existing concepts of the injury as a transient vascular disorder were no longer adequate and had to be revised to include a severe necrotizing effect.

Continuing their studies with this model (41, 42), they described in association with the injury a severe irreversible disruption of the circulation of the cord, immediate in onset, and characterized by an abrupt arrest of the toxic injection mass, an immediate impairment of vascular permeability barriers, torpid blood flow with development of severe sludging and a persistent elevation of vascular resistance in the area of injury. The changes took place in the absence of demonstrable vasospasm. These disturbances in vasomotion were so severe as to constitute, in their opinion, a formidable obstacle to any therapeutic

Table 2

Potentialization by Levophed of neurotoxic action of fixed volume intra aortic dose of varying concentrations of Hypaque, Angio Conray and Conray

First injection 2 ml/kg	Second injection 2 ml/kg	No. of dogs	Posture	Con- vul- sive re- sponse	Neurologic deficit	Anatomic lesions graded	Deaths
Agent	Dose mg	Concen- tration (%)			Agg R's	None Mild Mod Severe	0 1 2 3 4 5
Levophed 1		90	Supine	5	5		2 2 1
Levophed 1		100	"	1	4	5	1 4
Levophed 1		75	"	1	4	5	3
Levophed 1		50	"	4	2	1 1 3	2 1 2 1
Levophed 1		40	"	6	1	2 2	1 1 1
Levophed 1	Hypaque	35	"	5	5		3
Levophed 1		90	Prone	5	5		4 1
Levophed 1		90	"	1	4	5	1 3 1
Levophed 1		75	"	4	2	1 1 1 3	1 1 1 1
Levophed 1		50	"	4	1	4	1 2 1 1
Levophed 1		40	"	5	5		2 3
Levophed 1		100	Supine	5	1	6	4 2
Levophed 1		80	"	1	4	5	1 4
Levophed 1	Angio Conray	50	"	1	4	5	3 1
Levophed 1		40	"	5	5	5	2 1 2
Levophed 1		30	"	5	4	1	4 1
Levophed 1		60	"	5	5	5	1 4
Levophed 1	Conray	60	"	5	5	3	1 4
Levophed 1		50	"	5	2	3	1 3
Levophed 1		40	"	5	5	5	

Admission sed 30 sec after 1st injection

Death occurred within 8 hrs after administration of contrast agent

Conray Na iothalamate 80% and Conray (methylglucamine iothalamate 60%) In these tests the agents were injected manually or by an air-driven syringe via a retrograde femoral catheter passed into the abdominal aorta to the level of the 2nd lumbar vertebral body at a rate of 5 ml per sec. Two programs of administration of contrast agents were used (Tables 1-3). In one the volume dose was maintained at 2 ml/kg body weight and the concentration of each agent was progressively lowered from the highest level available. In the other the volume dose was progressively reduced by decrements of 50% and the concentration of each agent was maintained. In each program two test series were performed. In the first the contrast agent was administered without a preparatory injection. In the second the contrast agent was preceded 30 seconds by an injection of 1 mg of levarterenol (Levophed).

Table 1

Potentiation by Levophed of neurotoxic action of fixed volume intra aortic doses of varying concentrations of Urokon

First injection 2 ml/kg		Second injection ¹ 2 ml/kg		No of dogs	Posture	Con vul sive res ponse	Neurologic deficit	Anatomic lesion grade						Dea
Agent	Dose mg or con centra tion	Agent	Con centra tion % or dose					0	1	2	3	4	5	
						Neg Pos None Mild Mod Severe								
		Urokon	70	8	Supine	■		7				4	3	1
		Urokon	50	5	»	5		4				3	2	
		Urokon	40	5	»	1 4 1		4		1	2			
		Urokon	35	6	»	6			3	2				1
Ievophed	1	Urokon	50	5	»	3 2		5				1	3	1
Ievophed	1	Urokon	40	5	»	2 3		3				1	2	2
Ievophed	1	Urokon	35	5	»	2 3		5				3	2	
Urokon	35 %	Levophed	1 mg	5	»	5	4	1	2	2	1			
Levophed	1	Urokon	25	7	»	2 5		1 2 4			1	2	2	2
Ievophed	1	Urokon	20	5	»	4 1 4	1		1	1	3			
Levophed	1	Urokon	15	5	»	5			2	2	1			
		Urokon	70	7	Prone	3 2 3	3		1	2	3			1
		Urokon	50	5	»	5	5		2	1	2			
		Urokon	35	5	»	5	5		4	1				
Ievophed	1	Urokon	70	7	»	5 2		6				2	4	1
Ievophed	1	Urokon	35	7	»	3 2		5			2		3	
Urokon	35 %	Levophed	1 mg	5	»	5			2	3				
Ievophed	1	Urokon	25	5	»	5	2	3	1	1		1	2	
Ievophed	1	Urokon	20	5	»	5	5		3	2				

¹ Administered 30 sec after 1st injection

² Death occurred within 8 hrs after administration of contrast agent

agents — would be obtained in this experimental model if a vasopressor agent were used instead of vasodepressor drugs. The present report describes results of studies testing this postulate, and further exploration of the pathogenesis and therapeutics of contrast agent injury.

Experimental procedure

The procedure used followed the same plan reported previously (24-63) using the canine abdominal aortography model with the spinal cord as the principal test site. Four contrast agents were used in commercially available concentrations; namely Urokon (Na acetrizoate 70 %) Hypaque (Na [30 %] and methylglucamine [60 %] diatrizoate 90 %) Angio-

Table 2

Potentialization by *Levophed* of neurotoxic action of fixed volume intra aortic doses of varying concentrations of *Hypaque*, *Angio Conray* and *Conray*

First injection 2 ml/kg		Second injection 2 ml/kg		No of dogs	Posture	Con- vul- sive res- ponse	Neurologic deficit						Anatomic lesion grade						Died ^a
Agent	Dose mg	Agent	Concen- tration (%)				New	Pos	Ne	Mild	Not	Severe	0	1	2	3	4	5	
<i>Levophed</i>	1		90	5	Supine	5	5						2	2	1				
<i>Levophed</i>	1		90	5	"	1	4					5					1	4	
<i>Levophed</i>	1		75	5	"	1	4					5					2	3	
<i>Levophed</i>	1		50	6	"	4	2		1	1	3					2	1	2	1
<i>Levophed</i>	1		40	6	"	6		1	2		2					1	3	1	1
<i>Levophed</i>	1	<i>Hypaque</i>	30	5	"	5		5						2	3				
<i>Levophed</i>	1		90	5	Prone	5	5						4	1					
<i>Levophed</i>	1		90	5	"	1	4					5				1	3	1	
<i>Levophed</i>	1		75	6	"	4	2	1	1	1	3			1	1	2	2		
<i>Levophed</i>	1		50	5	"	4	1	4			1	2	1	2					
<i>Levophed</i>	1		40	5	"	5		5					2	3					
<i>Levophed</i>	1		80	6	Supine	5	1	6						4	2				
<i>Levophed</i>	1		80	5	"	1	4					5					1	4	
<i>Levophed</i>	1	<i>Angio- Conray</i>	50	5	"	1	4					5					3	2	
<i>Levophed</i>	1		40	5	"	5						5				2	1	2	
<i>Levophed</i>	1		30	5	"	5		4	1						1				
<i>Levophed</i>	1		60	5	"	5		5					4						
<i>Levophed</i>	1		60	5	"	5		5				3							
<i>Levophed</i>	1	<i>Conray</i>	60	5	"	5				2	3				1	4			
<i>Levophed</i>	1		30	5	"	5		2			1			2		3			
<i>Levophed</i>	1		40	5	"	5		5				5							

^a Died 15 and 30 sec after 1st injection

Death occurred within 8 hrs after administration of contrast agent

Conray Na iothalamate 80% and *Conray* (methylglucamine iothalamate 60%) In these tests the agents were injected manually or by an air-driven syringe via a retrograde femoral catheter passed into the abdominal aorta to the level of the 2nd lumbar vertebral body at a rate of 5 ml per sec. Two programs of administration of contrast agents were used (Tables 1-3). In one the volume dose was maintained at 2 ml/kg body weight and the concentration of each agent was progressively lowered from the highest level available. In the other the volume dose was progressively reduced by decrements of 50% and the concentration of each agent was maintained. In each program two test series were performed. In the first the contrast agent was administered without a preparatory injection. In the second the contrast agent was preceded 30 seconds by an injection of 1 mg of levarterenol (*Levophed*)

Table 3

Potentiation by Levophed of neurotoxic action of varied volume intra aortic doses of concentrated solutions of Urokon Hypaque Angio Conray and Conray

First injection		Second injection ¹		No of dogs	Posture	Con vul sive res ponse	Neurologic deficit					Anatomic lesion grade					Total
Agent	Dose mg	Agent	Dose ml/kg ²				Conc (%)	Dose ml/kg	0 1 2 3 4 5								
							Neg	Pos	None	Mild	Mod	Severe					
		Urokon	70 1	8	Supine	7									2	1	4
		Urokon	70 0.5	6	»	1	5	1	2				3	1	1	1	
		Urokon	70 0.25	5	»	3	2	3	1				1	1	2	1	
Levophed 1	0.25	Urokon	70 0.25	5	»	1	4						5			1	4
Levophed 1	0.125	Urokon	70 0.125	5	»	3	2	1					4	1		1	1
Levophed 1	0.06	Urokon	70 0.06	5	»	5		1	1	1			1		3		1
Levophed 1	0.5	Hypaque	90 0.5	6	»	2	4						5			2	3
Levophed 1	0.25	Hypaque	90 0.25	5	»	4	1		1				4		1		3
Levophed 1	0.125	Hypaque	90 0.125	5	»	5		4					1	4			1
Levophed 1	0.25	Angio	80 0.25	5	»								2	3			2
Levophed 1	0.125	Conray	80 0.125	5	»	5		3		1	1		3		1		1
Levophed 1	1.0	Conray	60 1.0	5	»	5		5					5				

Administered 30 sec after 1st injection

¹ Death occurred within 11 hrs following administration of contrast agent

² Volume dose = 1 ml or more

diluted to an equivolume dose and administered via the same route and at the same rate. The route of the preparatory injection coupled with the interval between it and the administration of the contrast agent was chosen 1) to expose the target vascular bed to the first arterial circuit of the vasoactive agent hence to its maximal influence and 2) to allow time for its systemic action to be initiated. At the end of the preparatory injection the catheter was emptied by flushing and was refilled with saline. A single test was carried out on each animal.

As in previous experiments control studies were used with the order of injection of test substances reversed the vasoactive agent following the contrast material. In these studies the primary objective was to observe the influence of a vasopressor agent upon the neurotoxic action of contrast agents. Comparison of relative neurotoxicity of the various media used was but an incidental feature of the study. Accordingly in the titrations of progressively reduced

concentrations in one series and volume doses in the other series these decrements were of smaller order in tests with Urokon and Hypaque agents used earlier in the study than with Angio-Conray and Conray which were tested later. Overly healthy adult mongrel dogs of both sexes weighing 6 to 20 kg were used. Anesthesia was induced by intravenous pentobarbital or by fluothane inhalation. With each injection the immediate neurologic and cardio-respiratory responses were noted. In selected animals arterial pressure changes were recorded on a Honeywell Visicorder. Upon recovery from the anesthesia and daily thereafter observations of the neurologic status were made. After a period usually of 3 to 7 days the animals were sacrificed by an overdose of nembutal for pathologic study after the manner previously reported (39). The cord was removed, fixed in neutral formalin and sectioned transversely at 0.3 to 0.5 cm intervals. The gross features of the cord were diagrammed on a model recording the transverse and longitudinal extent of the lesions. Confirmation of the gross observations was made with study of selected microscopic sections six or more for each specimen. Although the pathologic studies were focused primarily upon neurotoxic effects of the contrast agents with the spinal cord as the target site, gross surveys were made of somatic organs for overt damage and histological examinations of the kidneys were done on all the test animals. Hematoxylin-eosin stains were routinely used.

The immediate neurologic response, the post-injection neurologic status and the resultant anatomic changes in the spinal cord were classified by a grading system established in earlier studies as reported below.

Convulsion response This was graded as negative or positive according to these criteria:

Negative — No convulsion is recognized.

1. No response.

2. Brief (10 sec or less) feeble flexion and/or extension of the trunk and/or hind legs and tail without subsequent signs or abnormal irritability or changes in the reflexes.

Positive — Convulsive response occurs.

1. Prolonged (usually 20 to 50 sec) tonic flexion and/or extension involving the trunk, hind limbs and tail and sometimes the forelimbs and neck without a recognizable clonic phase and followed by a 1 to 15 min period of hyperreflexia during which clonus may be elicited by tactile stimulation or

2. Prolonged (usually 20 to 50 sec) tonic flexion and/or extension involving the trunk, hind limbs and tail, sometimes forelimbs and neck followed by a clonic phase lasting from 30 sec to 5 min followed by severe hyperreflexia of 3 to 20 min duration.

Neurologic deficit

None — No alteration of function is observed.

Mild — A mild paresis of the hind legs is produced which is not apparent until the animal is exercised. This dysfunction usually disappears over the course of 3 to 5 days.

Moderate — Paraparesis is more marked than above and the animal may show weakness in the hind legs with impairment of locomotion which usually improves over the course of 3 to 5 days.

Severe — A paraplegia, usually spastic but sometimes flaccid, is present.

4. Lesions These were considered the most sensitive and definitive criteria of injury.

Grade 0 — No lesions are demonstrable.

Grade 1 — There are sparsely scattered microscopic foci of destruction affecting gray or white matter present in one or more sections of spinal cord.

Grade 2 — There are multiple small (1 to 2 mm) scattered foci of destruction of gray matter with or without involvement of white matter or spinal nerve roots distributed over 5 to 10 cm of spinal cord

Grade 3 — Extensive focal discrete or confluent zones of destruction of gray matter with or without involvement of white matter or spinal nerve roots extending over 5 to 10 cm of spinal cord are present

Grade 4 — Complete or nearly complete devastation of gray matter with or without involvement of white matter or spinal nerve roots involving from 10 to 18 cm of cord is observed

Grade 5 — There is complete or essentially complete transverse necrosis of the spinal cord often with extensive destruction of spinal nerve roots involving from 10 to 18 cm of cord

Results

The results of these studies are summarized in Tables 1 to 3 and Figs 1 to 5. At 2 ml/kg volume doses the concentrated solutions of these contrast agents, except for Urokon, were without overt neurotoxic action or were but minimally noxious, even for supinely positioned animals. Urokon, however (Tables 1 and 3, Figs 1 and 2) produced severe cord injury at concentrations down to 40%, with an abrupt fall off in toxicity at 35%. When administered to prone positioned animals its neurotoxic action was strikingly reduced, as exemplified by a comparison of the effects of the 50% concentration upon animals in these two postures. When concentration was maintained and volume dose progressively reduced Urokon retained its neurotoxic potential at low doses, producing frequent severe cord injury at 0.5 ml/kg and occasional severe injury at 0.25 ml/kg. When Levophed was administered directly prior to the contrast agents, without exception a striking potentiation of neurotoxicity was produced. At 2 ml/kg doses, Urokon retained its toxicity until the concentration was reduced below 25%, and the favourable influence of the prone posture was overcome. Further, incredibly small doses of 70% Urokon were rendered toxic, devastating spinal cord injury almost invariably resulting with 0.125 ml/kg doses and occasionally with 0.06 ml/kg doses. The less noxious contrast agent, Hypaque, in 2 ml/kg doses was rendered severely neurotoxic by Levophed at concentrations of 40% and above for the supinely positioned animal and at 75% with the dog in the prone position. 90% Hypaque retained this neurotoxic action at a volume dose of 0.25 ml/kg and was occasionally injurious at 0.125 ml/kg (Tables 2 and 3, Fig. 3). Similarly, Angio Conray, administered in 2 ml/kg doses following Levophed, was severely neurotoxic at 40% and above, 80% Angio Conray was unpredictably noxious at 0.25 and 0.125 ml/kg doses (Tables 2 and 3, Fig. 4).

Conray, administered in 2 ml/kg doses following Levophed exerted a variable and generally severe neurotoxic action at 60% concentration. At this volume dose the toxicity of this agent was reduced at 50% and eliminated at 40%.

Levophed Order of injection	Levophed concentration (%)	Posture of dog	Cord lesion (each section indicates one animal)				
—	50	Supine					
—	50	Prone					
—	40	Supine					
—	30	Supine					
1st	30	Supine					
2nd	30	Supine					
—	30	Prone					
1st	30	Prone					
1st	20	Supine					
1st	10	Supine					

Fig. 1. Potentiation by Levophed (1 mg) of neurotoxic action of fixed volume (2 ml/kg) intra-aortic doses of varying concentration of Levophed.

Grade 2 — There are multiple small (1 to 2 mm) scattered foci of destruction of gray matter with or without involvement of white matter or spinal nerve roots distributed over 5 to 10 cm of spinal cord

Grade 3 — Extensive focal discrete or confluent zones of destruction of gray matter with or without involvement of white matter or spinal nerve roots extending over 5 to 10 cm of spinal cord are present

Grade 4 — Complete or nearly complete, devastation of gray matter with or without involvement of white matter or spinal nerve roots involving from 10 to 18 cm of cord ■ observed

Grade 5 — There ■ complete or essentially complete transverse necrosis of the spinal cord often with extensive destruction of spinal nerve roots involving from 10 to 18 cm of cord

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Conray, administered in 2 ml/kg doses following Levophed, exerted a variable and generally severe neurotoxic action at 60 % concentration. At this volume dose the toxicity of this agent was reduced at 50 % and eliminated at 40 %.

Levophed Order of mg at in	Hypaque		Cord lesion (each section indicates one animal)				
	Concen	ml/kg					
	tration ()						
-	40	2					
1st	90	2					
1st	50	2					
1st	40	2					
1st	30	2					
1st	90	0.5					
1st	90	0.25					
1st	90	0.125					

Fig 3 Potentiation by Levophed (1 mg) of neurotoxic action of intra aortic Hypaque administered 15 min before, positioned dogs

were recorded exhibited a rise in blood pressure and widening of pulse pressure. Further, a pressor response to the subsequent contrast agent was sometimes observed. This latter reaction had not been encountered in previous studies with contrast agents where a mild depressor effect had been the common observation (42). The varying pressure responses to contrast agents with and without vasoactive drugs are summarized elsewhere (64).

Levophed Order of injection	Urokon ml/kg	Cord lesion (each section indicates one animal)				
—	1					
—	0.5					
	0.25					
1st	0.25					
1st	0.125					
1st	0.006					

Fig. 2. Potentiation by Levophed (1 mg) of neurotoxic action of varied volume intra aortic doses of 70% Urokon administered to supinely positioned animals.

concentration. Reduction of the volume dose of 60%. Contrary to 1 ml/kg virtually removed its toxic action.

On the other hand, when Levophed was administered following instead of preceding a contrast agent no potentiation of neurotoxic action occurred (Table 1, Fig. 1). Of 10 dogs to whom the sequence of 2 ml/kg of 35% Urokon then Levophed, was administered, none exhibited evidence of severe functional or anatomical lesions.

In this large series of animals none demonstrated gross alterations of the somatic organs. Mild degrees of kidney injury, consisting of a slight increase in glomerular permeability to proteins and rare foci of tubular necrosis were occasionally seen. These changes were not increased in severity in the animals to whom Levophed was administered. The injection of Levophed produced regularly an acceleration of heart rate, and the animals in which these variables


























Levophed Order of injection	Conray		Cord lesions (each section indicates one animal)				
	Concentration	ml/kg					
—	60	2					
1st	60	2					
1st	60	1					
1st	50	2					
1st	40	2					

Fig 5 Potentiation by Levophed (1 mg) of neurotoxic action of intra aortic Conray administered to serially positioned dogs

vascular bed of the spinal cord would be expected. With small volume doses however an unpredictable relationship between the injection mass and the variable and infrequent major arterial tributaries to the spinal cord can readily be understood. The inhibition of the toxic spinal convulsive reaction to contrast agents by Levophed (Tables 1 and 3) an agent which augments the neurotoxic action of these media points to a direct local depressant action upon the central nervous system by this drug strong enough to block a response which is not inhibited by inhalation anesthetics or by pentobarbital anesthesia.

These studies have demonstrated an impressive augmentation in the neurotoxic action of contrast media or from another viewpoint a striking increase in susceptibility of experimental animals to these agents — resulting from the action of vasopressor drug. Yet marked as these results are they present but one aspect of the modifying effects of pharmacologic agents. A better conception of the magnitude of these influences may be gained by a comparison of these observations with previous studies demonstrating the protective action































Evophed Order of Injection	Angio Conray		Cord lesion (each section indicates one animal)				
	Concen- tration (%)	ml/kg					
—	90	2					
1st	80	2					
1st	40	2					
1st	30	2					
1st	80	0.25					
1st	80	0.125					

Fig. 4 Potentiation by Evophed (1 mg) of neurotoxic action of intra aortic Angio Conray administered to supinely positioned dogs

Certain other features of these studies stand out. These include the abrupt demarcation between innocuous and severely toxic doses of contrast agents, the variability of the results observed in the low volume dose series, and the unreliability of the convulsive response as an indicator of neurotoxic action in the pharmacologically altered subjects. The sharp transition between toxic and non toxic doses is graphically illustrated in the figures (1 to 5). These findings are consistent with previous observations and can be explained, as before (42), by the microcirculatory changes occurring in conjunction with the toxic reaction. The variability of the results in the low volume dose studies (Tables 2 to 3) contrast strikingly with the consistency of the effects encountered in the large volume dose studies. In the latter series a uniform exposure of the


























Levophed Order of injection	Conray		Cord les on (each section indicates one animal)				
	Concen tration ()	ml/kg					
—	60	2					
1st	100	2					
1st	100	1					
1st	50	2					
1st	40	2					

Fig 5 Potentiation by Levophed (1 mg) of neurotoxic action of intra aortic Conray administered to supine positioned dogs

vascular bed of the spinal cord would be expected. With small volume doses however an unpredictable relationship between the injection mass and the variable and infrequent major arterial tributaries to the spinal cord can readily be understood. The inhibition of the toxic spinal convulsive reaction to contrast agents by Levophed (Tables 1 and 3) an agent which augments the neurotoxic action of these media points to a direct local depressant action upon the central nervous system by this drug strong enough to block a response which is not inhibited by inhalation anesthetics or by pentobarbital anesthesia.

These studies have demonstrated an impressive augmentation in the neurotoxic action of contrast media or from another viewpoint, a striking increase in susceptibility of experimental animals to these agents — resulting from the action of vasopressor drug. Yet marked as these results are they present but one aspect of the modifying effects of pharmacologic agents. A better conception of the magnitude of these influences may be gained by a comparison of these observations with previous studies demonstrating the protective action

of vasodepressor substances against contrast agent injury in this experimental roortography model. At one extreme, the highly toxic medium, Urokon, was rendered virtually innocuous, even at 2 ml/kg intra roortic doses of the 70 % concentration. At the other extreme, volume doses of 70 % Urokon as small as 0.125 ml/kg were made highly neurotoxic, as were concentrations as low as 25 % in 2 ml/kg doses. Hypaque 90 %, otherwise non toxic at 2 ml/kg doses, produced severe spinal cord injury at 0.25 ml/kg doses when administered during an active vasopressor stimulus. Angio Conray 80 %, ordinarily innocuous at 2 ml/kg doses, was variably toxic at 0.25 ml/kg doses when administered following Levophed. These influences are graphically depicted by a comparison of diagrams of paired experiments demonstrating, on the one hand a protective effect from a previously reported study (Fig. 6) and on the other, a potentiating action (Fig. 7).

The intensification of the toxic potential of Conray by Levophed was less dramatic than with the other contrast media, pointing to a greater margin of safety in the use of this contrast medium in diagnostic angiology, a property considered to be related to the use of methyl glucamine rather than sodium as the vehicle for this agent.

Discussion

By pharmacologic induction of altered reactivity of the vascular system, it has been possible, using a canine roortography model, to protect the spinal cord from injury by roentgen contrast agents and, conversely, to potentiate the neurotoxic action of these agents. The potentiating action of a vasopressor drug confirms the authors' predictions based upon observations of the protective effect of vasodepressor substances, and supplies a vital link in our knowledge of the nature of contrast medium injury (64). The import of these observations is emphasized by the broad differences in toxicities of various contrast agents under these modifying influences. The utterly small amounts of contrast media productive of neurotoxic effects in these studies (0.06 to 0.125 ml/kg or 1.2 to 2.5 ml for a 20 kg dog) adds further dimension to the concept of the potential toxicity of these agents. Nowhere in the experimental literature has so injurious an effect been produced on so diffuse a target by such inconsiderable quantities of contrast agents. The amounts stand in sharp contrast to the volume doses used in other meaningful studies of the toxicity of these substances. For example, KUTT and associates (28, 29, 45) exploring effects upon formed elements, proteins and electrolytes of the blood injected multiple intracrotid doses of 0.25 to 0.50 ml into rats reaching a total in excess of 6 % of the blood volume. AUSMAN et coll., studying the immediate toxic action of these media upon

PROPHYLAXIS OF CONTRAST MEDIUM INJURY

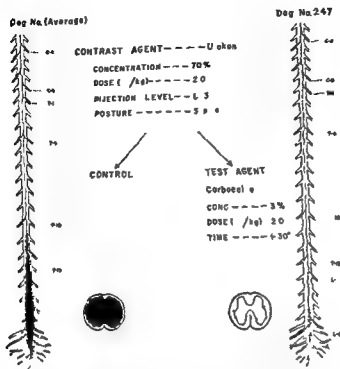


Fig. 6. This paired experiment from a previous study (64) demonstrates the protective action of a vasopressor drug against neurotoxic effects of roentgen contrast agents in the canine aortography model. A representative map of the arterial supply of the cord is presented rather than the actual pattern of blood supply for each animal. The dose of Urokon approximating 2/3 the MLD for the intra-aortic route is far in excess of clinically used doses and poses a most rigorous test of protective action. The blackened areas indicate the transverse and longitudinal extent of the spinal cord lesions produced in typical control animals (left) and the virtually complete prophylaxis of this neurotoxic effect achieved by the intra-aortic administration of carbochol 30 seconds prior to the injection of the contrast agent (right).

vascular endothelium injected quantities as high as 15 ml through the rat aorta. At the other extreme volume doses of concentrated agents far larger than those used in clinical aortography are converted into innocuous doses. An approximately 10 to 20 fold difference in toxicity of these substances for this experimental model has been demonstrated between the two extremes of modifying influences of vasoactive drugs. Additionally, all contrast agents used in this study have been demonstrated to have a toxic potential. In view

of vasodepressor substances against contrast agent injury in this experimental roentgenography model. At one extreme, the highly toxic medium, Urokon, was rendered virtually innocuous, even at 2 ml/kg intravascular doses of the 70 % concentration. At the other extreme, volume doses of 70 % Urokon as small as 0.125 ml/kg were made highly neurotoxic, as were concentrations as low as 25 % in 2 ml/kg doses. Hypaque 90 %, otherwise non toxic at 2 ml/kg doses, produced severe spinal cord injury at 0.25 ml/kg doses when administered during an active vasopressor stimulus. Angio Conray 80 %, ordinarily innocuous at 2 ml/kg doses, was variably toxic at 0.25 ml/kg doses when administered following Levo-phed. These influences are graphically depicted by a comparison of diagrams of paired experiments demonstrating, on the one hand a protective effect from a previously reported study (Fig. 6) and on the other, a potentiating action (Fig. 7).

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PROPHYLAXIS OF CONTRAST MEDIUM INJURY

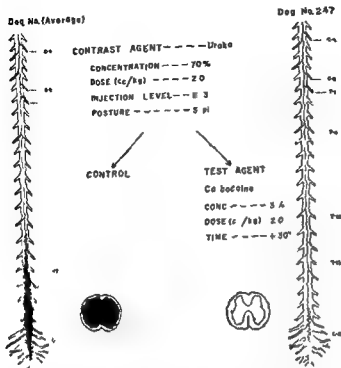


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vascular endothelium injected quantities as high as 15 ml through the rat aorta. At the other extreme volume doses of concentrated agents far larger than those used in clinical aortography are converted into innocuous doses. An approximately 10 to 20 fold difference in toxicity of these substances for this experimental model has been demonstrated between the two extremes of modifying influences of vasoactive drugs. Additionally all contrast agents used in this study have been demonstrated to have a toxic potential. In view

POTENTIATION OF CONTRAST MEDIUM INJURY

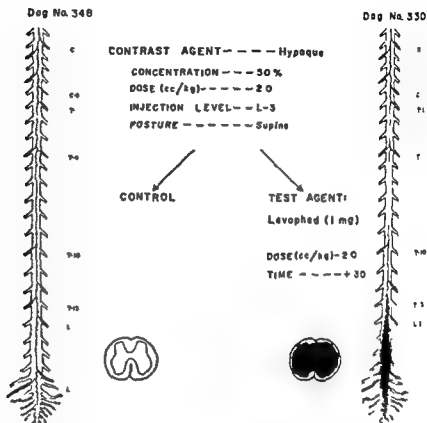


Fig. 7 This paired experiment demonstrates the potentiating action of a vasopressor drug upon neurotoxic effects of roentgen contrast agents in the canine roentgenography model. A representative map of the arterial supply of the cord is presented rather than the actual pattern of blood supply for individual animals. The spinal cord lesion resulting from the contrast agent is depicted by blackened areas in both longitudinal and transverse diagrams. The dose of Hypaque which is far below levels which have exhibited even minimal noxious effects (left) provides an unequivocal test of the ability of the vasopressor agent Levophed (levarterenol) to render this experimental model extremely susceptible to the neurotoxic action of contrast agents (right).

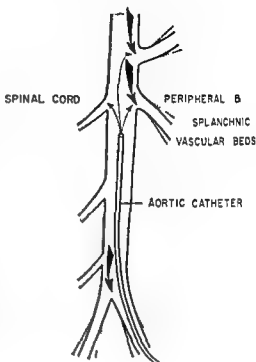
of these observations the possibility must be reconsidered that complications of carotid angiography may result from direct toxic action of the contrast agents rather than solely from faulty injection techniques and pre-existing vascular disease (52).

These modifying influences of vasoactive agents upon vulnerability of this experimental model to neurotoxic effects of contrast agents are explained, as before (64), on the basis of the differential reactivity of the vascular beds of the somatic organs and those of the nervous system. The latter have a notably

weaker reactivity to physical and to most chemical stimuli excepting the respiratory gases (32, 57). The stronger response of peripheral vessels to vasoactive agents could then override and reverse the primary reactions of the neural vascular bed. The vascular shunts resulting from these unequal responses to vasoactive agents are schematically presented in Figs 8 and 9. Injected at the height of the vasotonic response to levarterenol a contrast substance would be shunted toward the spinal cord because of the greater resistance to flow in the peripheral vessels. The possibility may be considered that these dramatic differences in neurotoxic reaction could signify that vasospasm played a major role in the production of the noxious action of contrast agents. Our earlier studies focused particularly upon the role of altered circulatory dynamics in the production of this injury have provided no evidence in support of such an interpretation. Rather a striking disturbance in vasomotion characterized by the circulatory events summarized above (42), occurred in the absence of demonstrable vasospasm. There is an abundant literature arguing against (6, 22, 23, 35, 36) the role of vasospasm in the production of contrast medium injury. One paper presents evidence that vasospasm exerts a protective effect (13). Urokon has been demonstrated to open up arteriovenous anastomoses in the extremities (56) and has even been used therapeutically to relieve vasospasm of peripheral vessels (37). Few reports of a vasospastic reaction to contrast agents are presented (8, 20, 47, 63). Our studies offer further argument against vasospasm being a factor in the production of injury based upon the absence of effects upon somatic organs paralleling those observed in the nervous system. If a vasospastic response to contrast agents represented the mechanism of production of toxic effects a severe injury to sites such as the kidney where the vascular bed is far more reactive than that of the central nervous system should theoretically have been produced by contrast agents administered at the height of a vasopressor response. For the same reason a direct potentiation of toxicity of contrast agents by levarterenol independent of alterations in vascular dynamics is considered unlikely. Parenthetically, the absence in these studies of injury to the kidney, an organ clearly in the potential target area during aortography (43, 50) indicates that a vascular shunt protecting the nervous system has not been created at the peril of important somatic organs. The decrease in neurotoxicity of an intra aortically administered contrast agent by turning an animal from the supine to the prone position (18, 39, 40, 60) an effect related to the differential gravitational flow of the iodinated medium rather than to altered circulatory dynamics (41, 42) — a protective effect which can be overcome, however, by the vasopressor shunt — emphasizes the lability and sensitivity of this concentration dependent experimental model.

VASODEPRESSOR SHUNT

Fig. 2 Schematic representation of the redistribution of blood flow produced by vasoactive agents as a result of the differential reactivity of the vascular beds of the somatic organs and those of the nervous system. Vasodepressor drugs produce a decreased vascular resistance in the somatic vascular bed diverting a potentially neurotoxic intra aortic injection mass to the periphery hence reducing it below noxious levels in the vascular bed of the spinal cord. This protective shunt has been induced by pharmacologic agents with diverse actions including local anesthetics, a ganglionic blocking drug and a smooth muscle relaxant. In view of the fact that the major changes in vascular tonus occur far distal to the depicted vascular beds this diagram errs on the side of simplicity in order to allow a presentation of blood flow. In actuality the order of vessels illustrated by this diagram may exhibit changes in caliber in an opposing direction i.e. a decrease in lumen diameter may occur in the face of peripheral vasodilatation.



Our studies present overwhelming evidence of a primary, concentration dependent, immediate, vasotoxic effect of contrast agents. This evidence is in agreement with earlier observations of BROMAN et coll. (10, 8) that concentration was a major factor in the noxious action of these substances. These workers observed that an agent of high concentration remaining in the cerebrovascular bed for but 1 second was more injurious than more dilute agents remaining in the vessels 10 to 70 seconds. KILLEN & OWENS (26), measuring renal oxygen availability after Urokon perfusion obtained findings supporting the interpretation that direct cellular toxicity rather than stress anoxia represented the mechanism of kidney injury by contrast media. Direct immediate injury to vascular endothelium has been produced by perfusions of large quantities of contrast media through vessels of small animals (1, 44, 46, 65) but the doses used are so large that it is difficult to apply these observations to clinical angiography. Thrombosis, the expected complication of severe injury to endothelium, is not a common feature of contrast medium reactions.

There is a growing body of literature, however, favoring the concept that the adverse effects of contrast media are of toxic ischemic character, secondary to action of these media on the blood plasma and its formed blood elements. Foremost among the toxic manifestations has been a transient

VASOPRESSOR SHUNT

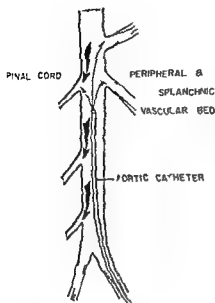
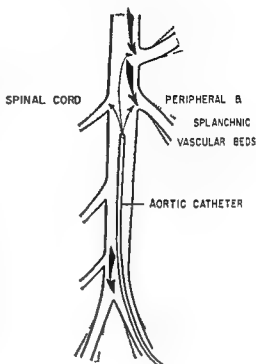


Fig. 9 Schematic representation of the redistribution of blood flow produced by vasoactive agents as a result of the differential reactivity of the vascular beds of the somatic organs and those of the nervous system. A vasopressor drug produces an increased vascular resistance in the somatic vascular bed diverting an otherwise innocuous intra aortic injection mass toward the vascular bed of the spinal cord hence augmenting its neurotoxic potential. An approximately 10 to 20 fold difference in toxicity of roentgen contrast agents has been demonstrated between the two extremes of modifying influences of vasoactive drugs in the canine aortography model. In view of the fact that the major changes in vascular tonus occur far distal to the depicted vascular beds this diagram errs on the side of simplicity in order to allow a presentation of the basis of redistribution of blood flow. Actually the order of vessels illustrated by this diagram may exhibit changes in caliber in an opposing direction i.e. an increase in lumen diameter may occur in the face of peripheral vasoconstriction.

phase of severe sludging (3, 4, 5, 21, 28, 29, 55) associated with acute pulmonary congestion, edema and hemorrhage, a disturbance of cardiac rhythm and observable microcirculatory changes explicable on the basis of temporary vascular obstruction (2, 3, 4, 5, 19). These observations have gained in importance with the demonstration that antisludging agents exert a protective action against large intravenous doses of contrast agents (3, 4, 5, 54) and to a lesser degree against smaller intra aortic doses (54). An increased resistance to blood flow in the limbs produced by intra arterial injection of a contrast agent has been linked with the presence of red blood cells in the perfusate (50). Further, renal damage from an intra arterial contrast agent has been prevented by eliminating red cells from the renal perfusate (12). Hypertonicity with the production of extra cellular hyperosmolarity, has been considered to be the cause of both the immediate and the delayed toxic effects of contrast agents particularly after large doses (16, 17, 48, 49). Certain contrast agents bind plasma albumin (30, 31) suggesting that protein binding may constitute an important mechanism of injury. The studies of HURT, McDOWELL and associates (28, 29, 45) demonstrating (1) a reversible aggregation of protein molecules leading to alteration in electrophoretic mobility of blood proteins and lipoproteins

VASODEPRESSOR SHUNT

Fig 8 Schematic representation of the redistribution of blood flow produced by vasoactive agents as a result of the differential reactivity of the vascular beds of the somatic organs and those of the nervous system. Vasodepressor drugs produce a decreased vascular resistance in the somatic vascular bed diverting a potentially neurotoxic intra aortic injection mass to the periphery hence reducing it below noxious levels in the vascular bed of the spinal cord. This protective shunt has been induced by pharmacologic agents with diverse actions including local analgesics, a ganglionic blocking drug and a smooth muscle relaxant. In view of the fact that the major changes in vascular tonus occur far distal to the depicted vascular beds, this diagram errs on the side of simplicity in order to allow a presentation of blood flow. In actuality the order of vessels illustrated by this diagram may exhibit changes in caliber in an opposing direction, i.e. a decrease in lumen diameter may occur in the face of peripheral vasodilatation.



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There is a growing body of literature, however, favoring the concept that the adverse effects of contrast media are of anoxic ischemic character, secondary to action of these media on the blood plasma and its formed blood elements. Foremost among the toxic manifestations has been a transient

towards greater safety particularly in pediatric angiology where the factor of dosage may constitute the principal hazard. If, as has been experimentally demonstrated, intrinsic vascular tonus is the major factor in the production of concentration dependent neurotoxic action, an approach is available towards the protection of the central nervous system from injury in aortography and angiocardiology. Considering that (1) it is in these procedures that the highest relative incidence of serious clinical complications have been encountered and (2) these injuries have commonly involved the spinal cord (25) brain stem and cerebrum regions outside the target zone of these procedures the potential value of this information is clearly evident. In the literature on diagnostic angiology frequent reference is made to the hazards of angiography to organs which have a pre-existing vascular disease (52). Our studies emphasize the alternate possibility, namely, that in the absence of overt brain disease the central nervous system may be imperiled by functional or anatomical vascular shunts resulting from the conditions leading to the need for diagnostic angiographic procedures. For example, this possibility can readily be envisaged in hypertension where there is a sustained elevation in systemic vascular tonus in occlusive aortic atherosclerosis where there is a strong collateral circulation via the important lumbar spinal radicles and in coarctation of the aorta with its multiple major collateral routes including the spinal branches of the intercostal arteries. These speculations are given reality by two reported instances of unilateral renal damage occurring from aortography of patients with hypertension secondary to unilateral renal vascular disease (43-50). In both of these cases the better functioning kidney was severely injured and the kidney with decreased function was protected, presumably because of its decreased blood flow. After these considerations it may be pertinent to ask whether the evidence should be re-examined that in the presence of cerebrovascular disease there are truly greater hazards to the central nervous system from four vessel angiography, a procedure which is atraumatic for the carotid vertebral system. To be specific, will cranial vascular stenosis render the brain less rather than more vulnerable to injury in angiography? To explore the questions raised by the observations recorded herein studies are being set up in our laboratories investigating the effectiveness of long acting pharmacologic agents and the influence of vascular shunts in modifying neurotoxic effects of contrast agents in the canine aortography model. If, as postulated, two mechanisms of injury exist, therapy effective against one may be ineffective against the other. Low molecular weight Dextran, valuable as it is in combating systemic microcirculatory changes produced by contrast agents, may have little influence upon a concentration dependent toxic action. Conversely, there is no theoretical basis on which to expect a vascular shunting action to protect from dose

(2) depression of free Ca and Mg levels leading to tetanic seizures and to disturbances of blood coagulation, (3) red cell aggregation and changes in viscomotion, point to important mechanisms in the production of contrast medium reactions and action on the blood and its formed elements, particularly when large doses of these agents are used.

To explain and to reconcile these conflicting sets of observations a unifying concept of the pathogenesis of contrast agent injury is proposed, namely, that there are two major mechanisms of injury. One is primary chemotoxic action exerted upon the vascular wall and parenchyma, the other is an ischemic hypoxic effect secondary to obstruction of the microcirculation. The primary toxic action is instantaneous in onset, and is concentration dependent. The secondary injury is predominantly dose dependent, and results from mass action upon the plasma and formed blood elements, with resultant microcirculatory disturbances leading to tissue anoxia. In any example of injury both mechanisms may participate, with one or the other playing the dominant role in production of damage.

The canine aortography model is presented as an experimental preparation providing a prime opportunity to study the pathogenesis of concentration dependent neurotoxic action of contrast agents. It is clearly evident from observations with this model, that the direction of shunting of an intra aortic injection mass, rather than quantity or initial concentration of the medium, is the major determinant of injury. The experimental studies of BERSTEIN & EVANS (3, 4, 5) focused upon acute cardiopulmonary effects of large intravenous doses of contrast agents represent a typical dose dependent model of injury, mediated through hypoxic ischemic sequelae of intravascular sludging. Consistent with this interpretation is the protective action exerted by the antisludging agent, low molecular weight Dextran, upon this latter model. Similarly, KUTT and associates (28, 29, 45), utilizing large quantities of contrast agents to explore effects upon blood protein, electrolytes and coagulation factors, make use of a dose dependent model. The studies of DEAN and associates (12), linking the presence of red cells in the renal vascular bed to vulnerability to toxic effects of a large dose (1 ml/kg) of 70 % Urokon argue against the concept advanced by the authors, but their experiments are complicated by their method of preparation of the target organ, namely the rapid injection of a massive quantity of perfusate (250 ml of 0.9 % saline in 90 sec) through an isolated aorticorenal segment.

Certain implications of importance to diagnostic angiology emerge from these studies. The development of a unified concept of contrast medium injury, allowing the delineation of dose dependent and concentration dependent toxic actions would greatly enhance the planning of angiographic procedures.

towards greater safety, particularly in pediatric angiology where the factor of dosage may constitute the principal hazard. If, as has been experimentally demonstrated, intrinsic vascular tonus is the major factor in the production of concentration dependent neurotoxic action, an approach is available towards the protection of the central nervous system from injury in aortography and angiocardiography. Considering that (1) it is in these procedures that the highest relative incidence of serious clinical complications have been encountered, and (2) these injuries have commonly involved the spinal cord (25), brain stem and cerebrum regions outside the target zone of these procedures the potential value of this information is clearly evident. In the literature on diagnostic angiology frequent reference is made to the hazards of angiography to organs which have a pre-existing vascular disease (52). Our studies emphasize the alternate possibility, namely that in the absence of overt brain disease the central nervous system may be imperiled by functional or anatomical vascular shunts resulting from the conditions leading to the need for diagnostic angiographic procedures. For example, this possibility can readily be envisaged in hypertension where there is a sustained elevation in systemic vascular tonus, in occlusive aortic atherosclerosis where there is a strong collateral circulation in the important lumbar spinal radicles, and in coarctation of the aorta with its multiple major collateral routes including the spinal branches of the intercostal arteries. These speculations are given reality by two reported instances of unilateral renal damage occurring from aortography of patients with hypertension secondary to unilateral renal vascular disease (43, 50). In both of these cases the better functioning kidney was severely injured and the kidney with decreased function was protected presumably because of its decreased blood flow. After these considerations it may be pertinent to ask whether the evidence should be re-examined that in the presence of cerebrovascular disease there are truly greater hazards to the central nervous system from four vessel angiography, a procedure which is atraumatic for the carotid vertebral system. To be specific, will cranial vascular stenosis render the brain less rather than more vulnerable to injury in angiography? To explore the questions raised by the observations recorded herein studies are being set up in our laboratories investigating the effectiveness of long acting pharmacologic agents and the influence of vascular shunts in modifying neurotoxic effects of contrast agents in the canine aortography model. If as postulated, two mechanisms of injury exist, therapy effective against one may be ineffective against the other. Low molecular weight Dextran valuable as it is in combating systemic microcirculatory changes produced by contrast agents may have little influence upon a concentration dependent toxic action. Conversely, there is no theoretical basis on which to expect a vascular shunting action to protect from dose

dependent systemic toxic effects of contrast agents. Tests of these latter questions are now under way in our laboratories.

The revival of the idea (1), originally advanced by BROMAN & OLSSON (8) that alterations in permeability of the blood brain barrier caused by contrast media might be exploited to increase the passage of therapeutic drugs into the brain causes some concern. When this consideration was first proposed, the neurotoxic potential of contrast media had not been fully appreciated. Observations that even the newer contrast media are capable of severe destructive action, and that the transition between non toxic and severely toxic doses of these agents is extremely abrupt, indicate that there is no clearly definable middle ground of relative safety within which borderline toxic action upon the cerebral vascular bed might be induced to provide a therapeutic advantage. Although appearing to be rather straightforward in our earlier experiments, the relationship between permeability changes and severity of a isotoxic action is clearly more complex than originally conceived. For example, Levophed, which potentiated the neurotoxic action of contrast agents, blocked convulsive response not suppressible by deep pentobarbital or fluothane anesthesia. The Levophed block may be explained on the basis of its depressant action upon the central nervous system (33, 34, 53). On the other hand, Papaverine and Arfonad, while offering strong protection from neurotoxic effect, demonstrated no ameliorating influence upon the convulsive response (64). On the basis of this observation attribution of the convulsive response to the direct irritative action of a contrast agent at the neuronal level consequent to damage of the blood brain barrier appears to be an oversimplified concept. In any event, a violent convulsive reaction does not always presage a necrotizing effect, nor does the lack of this discharge predicate the absence of toxic intratomic sequelae. Although changes in the blood brain barrier produced by contrast media have been demonstrated to be reversible (7, 9, 14, 15, 58, 59), correlative pathologic studies must be done to exclude the absence of irreversible intratomic effects in such experiments. In any event, at our present state of knowledge, the use of contrast agents for the purpose of increasing the passage of therapeutic drugs into the brain is not justified.

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SUMMARY

A vasopressor strongly potentiates and vasodepressors strikingly protect from neurotoxic effects of contrast media in an experimental aortography model. Dominance of somatic over neural vascular beds resulting in shunting explains these modifying influences. From these observations two mechanisms of injury are postulated: primary concentration dependent chemotoxic action upon tissue and dose dependent hypoxic ischemic effects secondary to microcirculatory obstruction. Of these effects only the former is considered amenable to action of vasoactive agents.

ZUSAMMENFASSUNG

Die neurotoxischen Wirkungen von Kontrastmitteln im experimentellen aortographischen Modellversuch werden von Vasopressoren potenziert, von Vasodepressoren hingegen verhindert. Die Erklärung dafür ist im Überwiegen der somatischen über die neuronalen Gefäßabschnitte — mit dem Resultat von Gefäßkurzschlüssen — zu suchen. Als Folge dieser Beobachtung kann man bei einer Schädigung zwei Mechanismen annehmen: Primäre Konzentrationsbedingte chemotoxische Gewebewirkung und dosisbedingte hypoxamische Wirkung, als Folge von Unterbrechung der Mikrozirkulation. Von diesen Reaktionen ist nur die erste für die Wirkung von vasoaktiven Stoffen als verantwortlich anzusehen.

RÉSUMÉ

Dans une experimentation aortographique les vasopresseurs potentialisent fortement et les vasodépresseurs empêchent de façon très nette les effets neurotoxiques des moyens de contraste. La dominance des réseaux vasculaires somatiques sur les réseaux vasculaires neuraux donne lieu à un shuntage explique ces influences modificatrices. Ces observations font supposer deux mécanismes lésionnels: l'un dépendant de la concentration est l'action chimiotoxique sur les tissus; l'autre dépendant de la dose est l'effet hypoxique ischémique du à l'obstruction microcirculatoire. De ces effets seul le premier est considéré comme sensible à l'action des agents vasoactifs.

REFERENCES

1. ALTMAN J I, YOUNG R and OWENS G. Radiopaque dyes and other agents in the production of endothelial damage. *J. surg. Res.* 4 (1964) 349.
2. ALSTON W F, WILCOX B R and BENDER H W. Experimental studies of the cardiovascular responses secondary to the injection of angiographic agents. *J. thorac. cardio. vasc. Surg.* 47 (1964) 283.
3. BERNSTEIN E F and EVANS R L. The protective action of low molecular weight dextran upon intravenous hypaque toxicity. *Surg. Forum* 11 (1960) 141.
4. — — Low molecular weight dextran. *J. Amer. med. Ass.* 174 (1960) 417.
5. — — PALMER J D, AARFANG T A and DAVIS R L. Studies of the toxicity of hypaque — 90 per cent following rapid intravenous injection. *Radiology* 76 (1961) 1111.
6. BLOOR B M, WRENCH F R JR and MARGOLIS G. Effect of intracarotid iodopyracet (diodrast) upon cerebral blood flow. *Arch. Neurol. Psychiat. (Chic.)* 71 (1954) 348.

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SUMMARY

A vasopressor strongly potentiates and vasod pressors strikingly protect from neurotoxic effects of contrast media in an experimental aortography model. Dominance of somatic over neural vascular beds resulting in shunting explains these modifying influences. From these observations two mechanisms of injury are postulated: primary concentration dependent chemotoxic action upon tissue and dose dependent hypoxic ischemic effects secondary to microcirculatory obstruction. Of these effects only the former is considered amenable to action of vasoactive agents.

ZUSAMMENFASSUNG

Die neurotoxischen Wirkungen von Kontrastmitteln im experimentellen aortographischen Modellversuch werden von Vasopressoren potenziert, von Vasodepressoren hingegen verhindert. Die Erklärung dafür ist im Überwiegen der somatischen über die neuralen Gefäßabschnitte — mit den Resultaten von Gefäßkurzschlüssen — zu suchen. Als Folge dieser Beobachtung kann man bei einer Schädigung zwei Mechanismen annehmen: Primäre konzentrationsbedingte chemotoxische Gewebswirkung und dosisbedingte hypoxämische Wirkung als Folge von Unterbrechung der Mikrozirkulation. Von diesen Reaktionen ist nur die erste für die Wirkung von vasoaktiven Stoffen als verantwortlich anzusehen.

RÉSUMÉ

Dans une expérimentation aortographique les vasopresseurs potentialisent fortement et les vasodépresseurs empêchent de façon très nette les effets neurotoxiques des moyens de contraste. La dominance des réseaux vasculaires somatiques sur les réseaux vasculaires neuraux donnant lieu à un shuntage explique ces influences modificatrices. Ces observations font supposer deux mécanismes lésionnels: l'un dépendant de la concentration est l'action chimiotoxique sur les tissus, l'autre dépendant de la dose est l'effet hypoxique ischémique dû à l'obstruction microcirculatoire. De ces effets seul le premier est considéré comme sensible à l'action des agents vasoactifs.

REFERENCES

1. ALTMAN J I, YOUNG R and OWENS G. Radiopaque dyes and other agents in the production of endothelial damage. *J. surg. Res.* 4 (1964) 349.
2. ALTMAN W I, WILCOX B H and BEYER H W. Experimental studies of the cardiovascular responses secondary to the injection of angiographic agents. *J. thorac. cardio-vasc. Surg.* 47 (1964) 283.
3. BERNSTEIN E F and ENAYAT R L. The protective action of low molecular weight dextran upon intravenous hypaque toxicity. *Surg. Forum* 11 (1960) 141.
4. — — Low molecular weight dextran. *J. Amer. med. Ass.* 174 (1960) 417.
5. — PALMER J D, AARFELT T A and DAVIS R L. Studies of the toxicity of hypaque — 90 per cent solution following rapid intravenous injection. *Radiology* 16 (1961) 88.
6. BLOOR B M, WRENCH F R JR and MARGOLIS C. Effect of intracarotid iodopyracet (diiodrast) upon cerebral blood flow. *Arch. Neurol. Psychiat. (Chic.)* 71 (1954) 348.

dependent systemic toxic effects of contrast agents. Tests of these latter questions are now under way in our laboratories.

The revival of the idea (1), originally advanced by BROMAN & OLSSON (8) that alterations in permeability of the blood brain barrier caused by contrast media might be exploited to increase the passage of therapeutic drugs into the brain causes some concern. When this consideration was first proposed, the neurotoxic potential of contrast media had not been fully appreciated. Observations that even the newer contrast media are capable of severe destructive action, and that the transition between non toxic and severely toxic doses of these agents is extremely abrupt, indicate that there is no clearly definable middle ground of relative safety within which borderline toxic action upon the cerebral vascular bed might be induced to provide a therapeutic advantage. Although appearing to be rather straightforward in our earlier experiments, the relationship between permeability changes and severity of vasotoxic action is clearly more complex than originally conceived. For example, Levophed, which potentiated the neurotoxic action of contrast agents, blocked convulsive response not suppressible by deep pentobarbital or fluothane anesthesia. The Levophed block may be explained on the basis of its depressant action upon the central nervous system (33, 34, 53). On the other hand, Papaverine and Arfonad, while offering strong protection from neurotoxic effect, demonstrated no ameliorating influence upon the convulsive response (64). On the basis of this observation attribution of the convulsive response to the direct irritative action of a contrast agent at the neuronal level consequent to damage of the blood brain barrier appears to be an oversimplified concept. In any event, a violent convulsive reaction does not always presage a necrotizing effect, nor does the lack of this discharge predicate the absence of toxic anatomic sequelae. Although changes in the blood brain barrier produced by contrast media have been demonstrated to be reversible (7, 9, 14, 15, 58, 59), correlative pathologic studies must be done to exclude the absence of irreversible anatomic effects in such experiments. In any event, at our present state of knowledge, the use of contrast agents for the purpose of increasing the passage of therapeutic drugs into the brain is not justified.

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- 28 — MILHORAT T H and McDOWELL F The effect of iodized contrast media upon blood proteins electrolytes and red cells *Neurology* 13 (1963) 492
- 29 KUTT H and McDOWELL F Effects of iodized contrast media upon electrophoretic mobilities of blood proteins and lipoproteins *J Lab and Clin Med* 59 (1962) 118
- 30 LASER E C ELIZONDO MARTEL G and GRANKE R C Potentiation of pentobarbital anesthesia by competitive protein binding *Anesthesiology* 24 (1963) 665
- 31 — — — The roentgen contrast media potentiation of nembutal anesthesia in rats *Amer J Roentgenol* 91 (1964) 153
- 32 LAZORTIES G *Vascularisation et circulation cerebrales* Masson & Cie Paris 1961
- 33 LEINDORFER A Analgesia and anesthesia induced by epinephrine *Amer J Physiol* 157 (1949) 116
- 34 — The action of sympathomimetic amines on the central nervous system and the blood sugar Relation of chemical structure to mechanism of action *J Pharmacol exp Ther* 98 (1950) 67
- 35 LINDREN P and TORNELL G Blood circulation during and after peripheral arteriography Experimental study of the effects of triurol (sodium acetrizoate) and hypaque (sodium diatrizoate) *Acta radiol* 49 (1958) 475
- 36 — Blood pressure and heart rate responses in carotid angiography with sodium acetrizoate (triurol) An experimental study in cats *Acta radiol* 50 (1958) 160
- 37 — NORDENSTROM B and TORNELL G Arterial spasm treated with sodium acetrizoate *Acta chir scand Suppl* 245 (1959) 351
- 38 MARCOLIS G The effect of various agents on the toxic action of contrast media Fourth Princeton Conference on Cerebrovascular Disease January 1964
- 39 — TARAZI A K and GRINSON K S Contrast medium injury to the spinal cord produced by aortography Pathologic anatomy of the experimental lesion *J Neurosurg* 13 (1956) 349
- 40 — et coll Evaluation of roentgen contrast agents used in cerebral arteriography I A simple screening method *J Neurosurg* 15 (1958) 30
- 41 — et coll Circulatory dynamics of the canine spinal cord Temporal phases of blood flow measured by fluorescein and scintiscintigraphic methods *J Neurosurg* 14 (1957) 506
- 42 — et coll Contrast medium injury to the spinal cord Role of altered circulatory dynamics *J Neurosurg* 16 (1959) 390
- 43 McCALLISTER B D HUNT J C and KINCAID O W Unilateral renal atrophy subsequent to renal arteriography *Proc Mayo Clin* 37 (1962) 523
- 44 McCONYELL F and MERSEREAU W A The effect of angiographic contrast media on arterial endothelium — an experimental study *J Canad Ass Radiol* 15 (1964) 14
- 45 McDOWELL F and BANG N U Discussion on complications of angiography Fourth Princeton Conference on Cerebrovascular Disease January 1964
- 46 MERSEREAU W A and ROBERTSON H R Observations on venous endothelial injury following the injection of various radiographic contrast media in the rat *J Neurosurg* 18 (1961) 789
- 47 RAYBOR R B and ROSS G Arteriography and vasospasm The effects of intracarotid contrast media on vasospasm *J Neurosurg* 17 (1960) 1075
- 48 READ R C Cause of death in cardioangiography *J thorac cardiovasc Surg* 38 (1959) 683
- 49 — JOHNSON J A VICK J A and MEYER M W Vascular effects of hypertonic solutions *Circulat Res* 8 (1960) 538

- 7 BROMAN T and OLSSON O The tolerance of cerebral blood vessels to a contrast medium of the diodrast group—an experimental study of the effect on the blood brain barrier *Acta radiol* 30 (1948) 326
- 8 — — Experimental study of contrast media for cerebral angiography with reference to possible injurious effects on the cerebral blood vessels *Acta radiol* 31 (1949) 371
- 9 — — Technique for the pharmacodynamic investigation of contrast media for cerebral angiography. Effect on the blood brain barrier in animal experiments *Acta radiol* 45 (1956) 96
- 10 — FORSSMAN B and OLSSON O Further experimental investigations of injuries from contrast media in cerebral angiography—summation of various injurious factors *Acta radiol* 34 (1950) 135
- 11 CANOSSO G BRUNELLI B CORTESI N and CARPI A Rapid serial angiography in the investigation of the pharmacology of brain circulation in the dog *Circulat Res* 10 (1962) 259
- 12 DEAN R E ANDREWS J H and READ R C The red cell factor in renal damage from angiographic media *J Amer med Ass* 187 (1964) 27
- 13 LOITZ E L THOMAS L B and WARD A A JR The effects of intracarotid diodrast *J Neurosurg* 9 (1952) 68
- 14 FLODMARK S and STEINWALL O A method for study of the interrelation between EEG and blood brain barrier phenomena *Acta physiol scand* 56 (1962) 112
- 15 — — Reversible blood brain barrier alteration induced by certain organic acids and indicated by means of EEG and dye tests *Acta physiol scand* 58 (1963) 368
- 16 CARBER G L and READ R C Protective effect of hypervolemia in cardioangiography *J Amer med Ass* 180 (1962) 376
- 17 CIAMMONA S I LURIE P R and SECAR W T Hypertonicity following selective angiocardiology *Circulation* 28 (1963) 1096
- 18 HOL R and SAKRAVEN O Spinal cord damage in abdominal aortography *Acta radiol* 42 (1954) 276
- 19 HOFF J O Some pharmacological aspects of radiopaque compounds *Ann N Y Acad Sci* 78 (1959) 727
- 20 INCYAR D H and SODERBERG V Cerebral vasomotor tone and EEG during injection of umbradil. An experimental study with a new method *Acta radiol* 47 (1957) 185
- 21 JOHNSON J H and KNESEY M H Intravascular agglutination of the flowing blood following the injection of radiopaque contrast media *Neurology* 12 (1962) 560
- 22 KÄSTROM T LINDGREN P and TORNELL G Changes in cerebral circulation during carotid angiography with sodium acetrizoate (triuro) and sodium diatrizoate (Hypaque) *Acta radiol* 50 (1958) 151
- 23 — — — Circulatory disturbances during cerebral angiography. An experimental evaluation of certain contrast media *Acta radiol* 54 (1960) 3
- 24 KENNAN P D TINDALL C I MARGOLIS G and WOOD R S The prevention of experimental contrast medium injury to the nervous system *J Neurosurg* 15 (1958) 92
- 25 KILLEN D A and FOSTER J H Spinal cord injury as a complication of aortography *Ann Surg* 152 (1960) 211
- 26 — and LANGE T M Investigation of means to prevent spinal cord and renal damage incident to urokon aortography *Surgery* 51 (1962) 338
- 27 — and OWENS G Changes of renal oxygen availability during Urokon and Hypaque aortography *Ann Surg* 152 (1960) 957

PETECHIAL REACTIONS IN ANGIOGRAPHY

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More careful examination of two other patients investigated with carotid angiography the same day revealed petechial hemorrhages of a much milder degree. Following this initial observation careful examination of every patient undergoing angiography carotid or otherwise was performed. This report is concerned with the incidence of this complication and one of its probable mechanisms.

Many complications secondary to cerebral angiography have been described in the literature but the local petechial reaction has received scant attention. A review of the American literature since 1950 reveals 18 cases of petechial hemorrhages as a complication of angiography (ECKER 1951, UHLEIN 1951, ABBOTT GAY & GOODALL 1952, DUNN, UHLEIN & HOLMAN 1956, DE SAUSSURE & KEIRNS 1962, SPUDIS, SCHARY, J. ALEXANDER & MARTIN 1962, LANG 1963, DAROFF & McENTEE 1964). Not included in these 18 are four cases reported by DAROFF & McENTEE (1964) which are part of this present series. ECKER (1951) has an illustration of a patient who underwent right cerebral angiography and had petechiae in the distribution of the right external carotid

- 50 SAKAMURA J A, ABBOT J P and NUNNALLY R M Unilateral malignant nephrosclerosis with superimposed unilateral cortical necrosis following an aortogram *Amer J clin Path* 35 (1961) 147
- 51 SCHINDT H W The behavior of the pial vessels during and after the intracarotid injection of roentgen contrast media *Acta radiol* 44 (1955) 100
- 52 SCHINDLER P and ZUNKER F Complications in direct percutaneous carotid arteriography *Arch Neurol* 11 (1963) 676
- 53 SCHWITZER A and WRIGHT S Action of adrenaline on the knee jerk *J Physiol* 88 (1937) 476
- 54 SESSIONS R T, KILLEN D A and LOSTER J H Low molecular weight dextran as a protective agent against the toxic effects of Urokon *Amer Surg* 28 (1962) 455
- 55 SOBIN S S, IRASHER W G, JACOBSON C and VAN ECKHOVEN F A Nature of adverse reactions to radiopaque agents *J Amer med Ass* 170 (1959) 1546
- 56 SAKO Y Hemodynamic changes during arteriography *J Amer med Ass* 183 (1963) 253
- 57 SOKOLOFF L Action of drugs on cerebral circulation *Pharmacol Rev* 11 (1959) 1
- 58 STEINWALL O An improved technique for testing the effect of contrast media and other substances on the blood brain barrier *Acta radiol* 49 (1958) 281
- 59 — Transport mechanisms in certain blood brain barrier phenomena — a hypothesis *Acta psychiatr Suppl* 314 1961
- 60 TARAZI A K, MARCOLIS G and GRIMSON K S Spinal cord lesions produced by aortography in dogs *Arch Surg* 72 (1956) 38
- 61 TINDALL G T and JACKSON J R Clinical evaluation of procaine and hypertonic glucose as possible adjuncts of carotid arteriography *J Neurosurg* 17 (1960) 43
- 62 — et coll Evaluation of roentgen contrast agents used in cerebral arteriography II Application of a new method *J Neurosurg* 15 (1958) 37
- 63 WIEDENMAN M P Distribution and sizes of vessels and their responses to intra arterial injections of radiopaque materials *Bibl Acta (Suppl Acta Anat)* 4 (1964) 156
- 64 YERASIMIDES T G, MARCOLIS C and PONTON H J Prophylaxis of experimental contrast medium injury to the spinal cord by vasodepressor drugs *Angiology* 14 (1963) 394
- 65 ZINNER G and COTTION R Morphologic changes in vessel endothelium caused by contrast media *Angiology* 10 (1959) 207

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- 52 SCHEINBERG P and ZUCKER F Complications in direct percutaneous carotid arteriography *Arch Neurol* 11 (1963) 676
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- 58 STEINWALL O An improved technique for testing the effect of contrast media and other substances on the blood brain barrier *Acta radiol* 49 (1958) 281
- 59 — Transport mechanisms in certain blood brain barrier phenomena — a hypothesis *Acta psychiat Suppl* 314 1961
- 60 TARAZI A K, MARGOLIS G and GRINSON K S Spinal cord lesions produced by aortography in dogs *Arch Surg* 72 (1956) 38
- 61 TINDALL C T and JACKSON J R Clinical evaluation of procaine and hypertonic glucose as possible adjuncts of carotid arteriography *J Neurosurg* 17 (1960) 43
- 62 — et coll Evaluation of roentgen contrast agents used in cerebral arteriography II Application of a new method *J Neurosurg* 15 (1958) 37
- 63 WIEDFMAN M P Distribution and sizes of vessels and their responses to intra arterial injections of radiopaque materials *Bibl Acta (Suppl Acta Anat)* 4 (1964) 756
- 64 YERASIMIDIS T G, MARGOLIS G and PONTON H J Prophylaxis of experimental contrast medium injury to the spinal cord by vasodepressor drugs *Angiology* 14 (1963) 394
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Fig. 1 Case 1 Bilateral facial and conjunctival petechial hemorrhages six hours after bilateral carotid angiography. Right side -- selective internal carotid angiography left side -- selective external and common carotid angiography. Petechiae are prominent in distribution of right external carotid artery.

artery and a transient left hemiplegia. UHLEIN (1951) described two cases of extensive petechial hemorrhages in a series of 100 angiograms utilizing Diodrast as the contrast medium. ABBOTT et coll (1952) found two cases of facial petechial hemorrhages in 174 angiograms and included in their report an additional case. In this particular article the patients demonstrated conjunctival, scleral and retinal petechiae. DUNN et coll (1956) found one case of petechial hemorrhage in 50 cerebral angiograms utilizing Hypaque. DE SAUSSURE (1962) described three cases of petechial hemorrhages in 300 angiograms utilizing a retrograde catheter technique, with the catheter in the right common carotid artery. SPUDIS et coll (1962) reported a case which came to postmortem examination while cutaneous petechiae were still present. In this instance no cerebral petechiae were seen. LANG (1963) shows a picture of extensive petechial hemorrhage following retrograde catheterization of the left subclavian artery and injection of Hypaque 50%. This is the only mention of petechial hemorrhages in his survey of complications of retrograde angiography encompassing 11 402 procedures. Finally, DAROFF & McENTEE (1964) describe



Fig 2 Case 2 Extensive right cutaneous petechiae over distribution of right external carotid artery. Sharp midline cut off. Patient also had a right visual scotoma.

six cases of petechial reactions following cerebral angiography with Hypaque 50%, and mention four additional cases from the present series. In none of the above reports was an etiologic mechanism demonstrated. In one of DAROFF & McENTEE's cases a postmortem examination was obtained six days after the angiography and no cerebral petechiae were noted. The cutaneous, mucosal and conjunctival petechiae had presumably disappeared since they had started to fade four days prior to death. The cause of death in this case was a ruptured aneurysm of the posterior communicating artery.

Case reports

Case 1 AC A 58-year-old white male admitted with right hemiparesis, right hemihypesthesia and involuntary movements of the right hand progressive for 11 months. A bilateral cerebral angiography was performed utilizing the percutaneous catheterization technique as

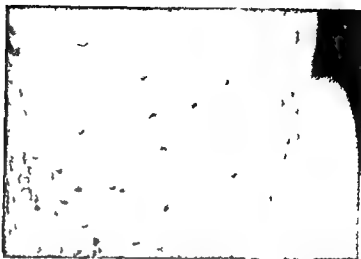


Fig. 3 Case 4. A moderate number of petechial hemorrhages over distribution of left external carotid artery. Close up view of the neck.

described by Seldinger (1953). Following puncture of the right common carotid artery the catheter was placed in the right internal carotid artery. Two injections of 7 ml Hypaque 50% were made. The attempt at selective catheterization of the right external carotid artery was unsuccessful. Puncture and catheterization of the left common artery was then performed with the tip of the catheter in the left external carotid artery. Two injections of 8 ml each of Hypaque 50% were made. Following this the catheter was withdrawn into the common carotid artery and 2 injections of 8 ml Hypaque were made. A left parasagittal meningioma was found.

Six hours after the angiography multiple petechiae were seen over the distribution of both external carotid arteries, being more prominent on the right (Fig. 1). These were entirely asymptomatic. It should be noted that no contrast material had been injected into the right external carotid artery but only into the internal carotid artery, yet there were decidedly more petechial hemorrhages on the right side of the face. These hemorrhages began to fade spontaneously 48 hours after the examination and no residua were noted.

Case 2 M A. A 40 year old white female entered the hospital for investigation of a possible intracranial aneurysm or vascular malformation. Percutaneous punctures of the left and right common carotid arteries were made (left side 2 injections of 8 ml Hypaque 50% and 1 injection of 6 ml; right side 2 injections of 8 ml Hypaque 50% and 5 injections of 6 ml). The findings were normal.

When seen approximately 5 hours after the examination many petechial hemorrhages of the face and neck in the distribution of the right external carotid artery were noted (Fig. 2). In addition hemorrhages were present in the conjunctival sac and on the hard palate on the right. The patient complained of a blank spot in her vision when she used her right eye and funduscopy revealed evidence of retinal hemorrhage. A coagulogram was entirely normal.

The petechial reaction began to fade spontaneously after approximately 24 hours and dis-



Fig 4 Starch granules stained with PAS 480

appeared in 3 days. Over the ensuing 3 months the scotoma decreased in size but a repeat evaluation of the visual fields demonstrated a persistent small scotoma on the right.

Case 1 HT. A 41-year-old Negro male was admitted for evaluation of blackout spells. Bilateral percutaneous carotid angiographies were performed and 8 ml Hypaque 50[®] were injected twice into each carotid artery. No abnormalities were seen on the films.

Approximately 5 hours after this examination petechiae were seen bilaterally in the conjunctivae and faintly over the distribution of the external carotid arteries. There were no subjective symptoms and the petechiae faded spontaneously over the next 3 days.

Case 4 MG. A 57-year-old white female was admitted with a diagnosis of stenosis of the proximal left internal carotid artery and underwent percutaneous left carotid angiography. Two injections of 8 ml Hypaque 50[®] were made.

Because of the findings in the three preceding cases the patient was seen 3 hours after angiography. No petechial hemorrhages were noted. She was revisited 14 hours after angiography and careful inspection demonstrated a moderate number of petechial hemorrhages on the left side of her face and neck (Fig 3). Although she had no subjective complaints, a right homonymous hemianopsia and a slight right hemiparesis were noted; these had not been present prior to angiography.

Two days following the angiography the stenotic left carotid artery was repaired and her postoperative course was unremarkable. The hemiparesis cleared but the homonymous hemianopsia persisted.

Table 1

Incidence of petechial hemorrhages

	No. of patients	No. of carotid angiographies	No. of cases with petechiae	% of cases with petechiae
With gloves	115	191	45	38
Without gloves	125	219	5	4

Table 2

Comparison of contrast material and patients with and without petechial reactions

Contrast medium	Presence of petechiae	Average age Male/female	No. of injections of contrast medium per angiography	Total volume of contrast medium per angiography	Duration of needle in situ	No. of patients
Hypaque 50 %	Yes	46 1/43 7	2 4	21.5 ml	42	14
Hypaque 50 %	No	42 1/41 6	2 9	22.9 ml	36.5	115
Conray	Yes	18 6/37 9	3 1	23.1 ml	31.5	27
Conray	No	17 4/47 7	2 9	23.3 ml	27	60
Renografin 60	Yes	41 5/13 3	2 8	24.5 ml	33	9
Renografin 60	No	47 5/48 7	3 4	27.2 ml	44	15

Investigation

Clinical As the four cases described above had occurred on the same day the entire angiographic procedure was reviewed the following day, and no change in the technique of preparing, cleaning, or sterilizing the instruments was elicited. Our own routine of angiography was carefully reviewed, and no breaks in technique could be detected. As the only other possible variable was the contrast medium utilized the entire remaining batch of material (Hypaque 50 %) was returned to the manufacturer for testing, the results of this analysis were entirely negative.

For empirical reasons it was decided to utilize other forms of contrast medium (Renografin 60, Conray, and in addition, in angiography of other areas of the body, Angio Conray and Cardiografin). At the same time detailed records were kept of each patient undergoing angiography, the patient being visited at least once four hours after the procedure.

Between March 1963 and September 1963, 172 carotid angiographies were performed in 105 patients, and follow up examinations demonstrated 38 patients with petechial hemorrhages. This yielded a 38 % incidence of



Fig 5 Microscopic section of rat's brain showing four intravascular starch granules $\times 170$

petechial hemorrhages among these patients. Most of these were confined to the conjunctiva and palate, were mostly bilateral and if they had not been searched for would almost certainly have gone unnoticed. An occasional case exhibited a moderate number of petechial hemorrhages but no patients in this series demonstrated the overwhelming reaction seen in cases 1 and 2 (Figs 1 and 2).

Because of the high incidence of petechial reactions a renewed search for an etiologic agent was made. When it was noticed that minute particles of powder were floating on the surface of the isotonic saline solution used to irrigate the needle or catheter while it was *in situ* it was decided that wherever feasible the procedure would be done without the use of gloves but following a surgical scrub of the hands. Since that time 219 carotid angiographies have been performed in 125 patients without the use of gloves. In this group five patients demonstrated between one and five petechiae in the conjunctiva. This yields an incidence of 4% of the patients. During this same period gloves were used in 19 angiographies on 10 patients and in 7 of these petechial reactions were seen. The compared results are shown in Table 1.

A detailed comparison was made of the contrast material used in patients

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Fig 5 Microscopic section of cat's brain showing four intravascular starch granules $\times 170$

petechial hemorrhages among these patients. Most of these were confined to the conjunctiva and palate were mostly bilateral and if they had not been searched for would almost certainly have gone unnoticed. An occasional case exhibited a moderate number of petechial hemorrhages but no patients in this series demonstrated the overwhelming reaction seen in cases 1 and 2 (Figs 1 and 2).

Because of the high incidence of petechial reactions a renewed search for an etiologic agent was made. When it was noticed that minute particles of powder were floating on the surface of the isotonic saline solution used to irrigate the needle or catheter while it was *in situ* it was decided that wherever feasible the procedure would be done without the use of gloves but following a surgical scrub of the hands. Since that time 219 carotid angiographies have been performed in 125 patients without the use of gloves. In this group five patients demonstrated between one and five petechiae in the conjunctiva. This yields an incidence of 4% of the patients. During this same period gloves were used in 19 angiographies on 10 patients and in 7 of these petechial reactions were seen. The compared results are shown in Table 1.

A detailed comparison was made of the contrast material used in patients



Fig. 6. Microscopic section of cat's brain showing perivascular petechial hemorrhage. 480

with and without petechiae is well as the age, sex, number of injections, total amount injected into each artery, and duration of examination. No difference was noted between the two groups, except for the presence or absence of gloves and therefore of powder (Table 2). The probability of the observed difference being due to a random sampling error is appreciably less than five in one thousand ($p < 0.005$).

During this same interval petechial hemorrhages were noted from time to time following roentgenographies and selective injections of major vessels arising from the aorta. Since all of these studies were done utilizing catheters, gloves were used in each instance.

Experimental. In order to test the relationship between the particulate matter and petechial hemorrhages, saline containing some of the powder was injected into the carotid arteries of cats.

The powder is a standard sterile material which is a reduced form of corn starch. It consists of amylose and amylopectin and contains 2% magnesium oxide as a filler. The particles range in size from 0.5 to 40 microns with an average of 25 to 30 microns. When introduced into the cavities of the body it is broken down enzymatically as a carbohydrate and absorbed in about seven days in the experimental animal. It is completely insoluble in water, saline or blood.

An attempt was made to equate the injections into the carotid artery of the cat with the equivalent injections into the human patients. Some of the powder

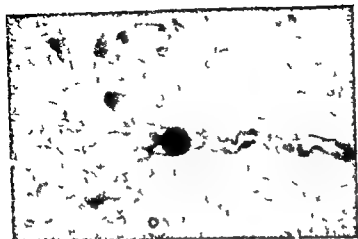


Fig 7 Microscopic section of cat's brain showing occlusion of small arteriole by starch granule $\times 480$

was sprinkled onto the surface of isotonic saline injected into the carotid artery of the cat and approximately two hours later the cat was sacrificed. In one case the vessels on the surface of the brain were observed during the injection and it could be seen that during the injection of saline the blood-filled vessels became transiently filled with saline and appeared as crystal clear tubes. During the injection of saline containing the powder suspension instead of the vessels refilling with blood after two or three seconds several of the vessels were seen to remain filled with saline for a period up to 20 minutes.

Fig 4 demonstrates a PAS stain of the starch granules and Fig 5 reveals a section of the cat's brain showing several of the granules lying within the blood vessels. Fig 6 under higher magnification demonstrates a typical perivascular petechial hemorrhage and Fig 7 also under higher magnification demonstrates a starch particle within a small arteriole.

Discussion

While these petechial reactions in the past have been characterized as benign and self-limited (1964) this has not been our experience. In case 2 of this report there must be a presumptive relationship between the angiography and the scotoma which accompanied the petechial reaction. In case 4 it is uncertain whether the homonymous hemianopsia is related to the angiography. It seems reasonable to assume, however, that during injections of the common carotid artery the bulk of the injection goes into the internal carotid tree since approximately two thirds of the blood follows this path. Any

particulate foreign matter would, statistically, be distributed likewise. Although serious sequelae have not been reported previously, it is quite reasonable to assume that multiple minute infarctions can occur throughout the cerebral hemispheres without producing neurologic deficits. It seems obvious, however, that any possible insult to the brain must be avoided. From the experience at our institution there is a definite relationship between the occurrence of petechial hemorrhages and the use of powder on the gloves as demonstrated in the clinical material and in the experimental material. The obvious solution is to keep the powder out of the material to be injected. This can be accomplished by using an entirely closed system as advocated by RINE (personal communication). However, we have noticed a tendency on the part of angiographers wearing gloves to be less careful about not handling the tip or shaft of the needle, another source of possible contamination.

The presence of the few petechial hemorrhages in the five patients in the series who were examined without gloves obviously cannot be explained on the basis of the powder. If the starch derivative powder, acting as particulate material, can produce this reaction there is no reason why other particulate material cannot do the same. SILBERMAN *et coll.* (1960) have reported five cases of foreign body emboli due to cotton fibers being introduced during cerebral angiography. CHASON *et coll.* (1963) reported five other cases of cotton fiber embolism, in addition to two cases of cotton fiber implantation into the carotid artery directly during cerebral angiography. COHEN *et coll.* (1964) have reported fat emboli in the retina following angiography; this was explained on the basis of atheromata being introduced into the blood stream at the site of puncture of the carotid artery. Either or both of these explanations may be involved in the five cases of petechial hemorrhages in our series in which gloves were not used.

BROMAN & OLSSON (1948, 1949) have previously demonstrated the effects of Diodrast on cerebral blood vessels in the rabbit. Using various concentrations of Diodrast injected over varying lengths of time, they demonstrated an increase in permeability of the cerebral vessels resulting in edemas, stasis, and punctate hemorrhages.

Therefore, it must be assumed that there is more than one mechanism involved in the production of these petechiae, but certainly particulate material must be included as one of these mechanisms.

Acknowledgements

I wish to express my appreciation to Dr. Nicholas Gonatas and Dr. Gabriel Schwarz of the Department of Neurology for their assistance with the microscopic sections and to Dr. G. Milton Shy and Dr. Eli Goldensohn of the Department of Neurology for the use of their animal laboratory.

SUMMARY

A discussion of the incidence and origin of petechial reactions associated with cerebral angiography is presented. Clinical and laboratory evidence indicated that the powder used on surgically sterile gloves is one of the causative factors.

ZUSAMMENFASSUNG

Das Auftreten und die Ursprung von petechialen Reaktionen im Zusammenhang mit cerebraler Angiographie wird besprochen. Klinische Untersuchungen und Laboratoriumsuntersuchungen haben ergeben, dass das für die chirurgischen sterilen Handschuhe verwendete Puder einer der ursächlichen Faktoren darstellt.

RESUMÉ

L'auteur étudie la fréquence et l'origine de pétéchies en relation avec l'angiographie cérébrale. Les recherches cliniques et de laboratoire ont montré que la poudre utilisée sur les gants chirurgicaux stériles est une des causes de cet incident.

REFERENCES

- ABBOTT K. H., GAY J. R. and GOODALL R. J. Clinical complications of cerebral angiography. *J. Neurosurg.* 9 (1952) 258.
- BROWN T. and OLSSON O. The tolerance of cerebral blood vessels to a contrast medium of the Diodrast group. An experimental study of the effect on the blood brain barrier. *Acta radiol.* 30 (1948) 326.
- — Experimental study of contrast media for cerebral angiography with reference to possible injurious effects on the cerebral blood vessels. *Acta radiol.* 31 (1949) 321.
- CHASON J. L., LANDERS J. W. and SWANSON R. E. Cotton fiber embolism: a frequent complication of cerebral angiography. *Neurology* 13 (1963) 558.
- COGAN C. G., KAWABARA T. and MOSER H. Fat emboli in retina following angiography. *Arch. Ophthalmol.* 71 (1964) 308.
- DAROFF R. B. and McENTEE W. J. Local petechial reactions following cerebral angiography with Hypaque. *J. Neurosurg.* 21 (1964) 381.
- DESJARDINS R. L. and KEIRNS M. M. Retrograde carotid catheterization for arteriography. *J. Neurosurg.* 19 (1962) 207.
- DUNN J. JR., LITTLE A. and HOLMAN C. B. The use of sodium diatrizoate in cerebral angiography. *J. Neurosurg.* 13 (1956) 677.
- ECKER A. The normal cerebral angiogram. First edition. Charles C. Thomas, Springfield Ill. 1951.
- LANGE K. Complications of retrograde percutaneous arteriography. *J. Urol.* 90 (1963) 604.
- RINE A. J. Personal communication.
- SEIDINGER S. I. Catheter replacement of the needle in percutaneous arteriography. A new technique. *Acta radiol.* 39 (1953) 368.
- SILBERMAN J., CRAVOTTO H. and FEIGEN I. Foreign body emboli following cerebral angiography. *Arch. Neurol.* 3 (1960) 711.
- SPEDIS E. A., SCHARY J. M., ALEXANDER E. and MARTIN J. F. Dissecting aneurysms in the head and neck. *Neurology* 12 (1962) 867.
- WILLIAMS A. Cerebral angiography. *Proc. Mayo Clin.* 26 (1951) 133.

particulate foreign matter would, statistically, be distributed likewise. Although serious sequelae have not been reported previously, it is quite reasonable to assume that multiple minute infarctions can occur throughout the cerebral hemispheres without producing neurologic deficits. It seems obvious, however, that any possible insult to the brain must be avoided. From the experience at our institution there is a definite relationship between the occurrence of petechial hemorrhages and the use of powder on the gloves as demonstrated in the clinical material and in the experimental animal. The obvious solution is to keep the powder out of the material to be injected. This can be accomplished by using an entirely closed system as advocated by Rine (personal communication). However, we have noticed a tendency on the part of angiographers wearing gloves to be less careful about not handling the tip or shaft of the needle, another source of possible contamination.

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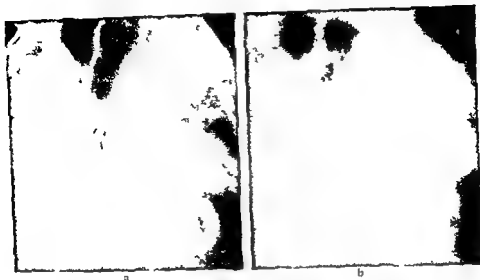


Fig 1 a) Full axial view left subclavian injection no compression. The basilar artery and its branches are seen. b) Right common carotid artery compression. Forward filling of the right posterior common carotid artery and filling of the right internal carotid artery.

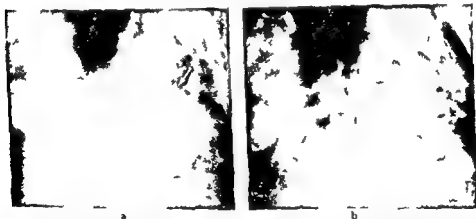


Fig 2 Full axial views left subclavian injection. a) Left common carotid artery compression. Forward filling of left internal carotid artery (arrow). b) Bilateral common carotid artery compression. Forward filling of both internal carotid arteries with filling of all the major arteries forming the circle of Willis.

possible that the right internal carotid tree has filled across the anterior part of the circle of Willis and not through the right posterior communicating artery. Previous tests with compression of the right carotid artery only will have shown patency or otherwise of the right posterior communicating artery.

ANGIOGRAPHIC DEMONSTRATION OF THE CIRCLE OF WILLIS

by

LEON MORRIS

At the last Symposium Neuroradiologicum, GRISLEPPE (1963) described compression tests in vertebral angiography to demonstrate the flow of contrast medium in the posterior part of the circle of Willis. Our experience has been largely in agreement with his assessment of these tests.

We have, however, modified the technique which in the past year we have carried out in the following manner. Indirect vertebral angiography is performed by subclavicular catheterisation of the subclavian artery. Wherever possible the left side is injected in order to avoid contamination of the intracranial circulation by contrast material injected into the innominate artery and thus into the right common carotid artery.

Films are taken with the head in the full neutral position. Four films are secured: 1) without carotid compression (Fig 1 a), 2) with compression of the right common carotid artery (Fig 1 b), 3) with compression of the left common carotid artery (Fig 2 a), and 4) with compression of both common carotid arteries (Fig 2 b).

These multiple compression tests are essential if one is to avoid any error in interpretation which may result from excessive cross flow in the anterior part of the circle of Willis. For example, in the case demonstrated in Fig 2 b, it is

artery. Occasionally, only needle puncture of the subclavian artery was possible and in these cases films were exposed in the half axial position.

A large enough series of cases of aneurysm treated by carotid occlusion has not been accumulated to assess the significance of our pre-operative compression tests but we feel that on the basis of these tests we can give some guidance to the surgeon as to the choice of treatment in each particular case. A very large series of cases with pre and post operative studies of the circle of Willis will be necessary before we can draw any valid conclusions concerning the role of the circle of Willis in maintaining blood flow in aneurysm treated by carotid occlusion methods.

Acknowledgement

I wish to record my indebtedness to Mr J. R. Gibbs of the South East Metropolitan Regional Neurosurgical Unit for his assistance and encouragement.

SUMMARY

In order to evaluate the circulation in the posterior part of the circle of Willis indirect vertebral angiography is performed and compression tests are carried out with the head in the full axial position. The method and results are described.

ZUSAMMENFASSUNG

Um die Zirkulation im rückwärtigen Teil des *circulus Willisii* zu erforschen wurde indirekte vertebrale Angiographie durchgeführt und Kompressionsteste in axialer Schädellage gemacht. Methode und Ergebnisse werden beschrieben.

RÉSUMÉ

Pour juger la circulation dans la partie postérieure du cercle de Willis l'auteur fait une angiographie vertébrale indirecte avec des épreuves de compression la tête étant en hyperextension. Il décrit sa méthode et ses résultats.

REFERENCE

- GRYSPEERDT G. L. Angiographic studies of the blood flow in the circle of Willis. *Acta radiol* Diagnosis 1 (1963) 298.



Fig 3 Half axial view left subclavian injection. Bilateral common carotid artery compression. All elements of the circle of Willis are seen with some superimposition of the vessels

Certain difficulties have been encountered with this technique

1 Failure to obtain a satisfactory subclavian catheterisation. In these cases, and in fact in many cases as a primary procedure, we now use the approach through the axillary artery. If necessary, the femoral approach could be used

2 If the right side is injected, one cannot assess accurately spontaneous forward flow in the right posterior communicating artery, because of simultaneous filling of the right common carotid artery

3 Failure to get satisfactory carotid compression

4 Failure to get a good full axial position of the head

If the failure is due to either of these last two factors, one must be satisfied to test the flow in the posterior communicating arteries with the head in the half axial position. This allows adequate examination of the flow in the circle of Willis, but excludes the clearer anatomical delineation of the full axial view. Fig 3 shows the result achieved with this method

In our small series of cases we have achieved a satisfactory examination in about 60 %. In most of the other cases we have obtained much of the information by modifying the procedure as described above. In the few cases where we were completely unsuccessful the reason was failure to fill the basilar artery because of a small vertebral artery or an anomalous origin of the vertebral

artery. Occasionally, only needle puncture of the subclavian artery was possible and in these cases films were exposed in the half axial position.

A large enough series of cases of aneurysm treated by carotid occlusion has not been accumulated to assess the significance of our pre-operative compression tests but we feel that on the basis of these tests we can give some guidance to the surgeon as to the choice of treatment in each particular case. A very large series of cases with pre- and post-operative studies of the circle of Willis will be necessary before we can draw any valid conclusions concerning the role of the circle of Willis in maintaining blood flow in aneurysm treated by carotid occlusion methods.

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VERTEBRAL ARTERIOVENOUS FISTULA COMPLICATING VERTEBRAL ANGIOGRAPHY

by

THOMAS H. NEWTON and JOHN DARROCH

The need for detailed vertebral angiography has increased with advances in neurosurgical techniques and many approaches have been described. Direct percutaneous puncture of the vertebral artery in the neck (LINDGREN 1950, MONES 1961, SUGAR, HOLDEN & POWELL 1949 and SUTTON 1962) is the most common method for vertebral angiography. The techniques as recommended by various authors have differed primarily in the level of arterial puncture and the type of needle used.

The most commonly reported complication of vertebral angiography is transient occipital lobe dysfunction with visual field defects (SILVERMAN, BERGMAN & BENDER 1961). Failures in technique have resulted in extravasation of contrast medium, hematoma, occlusion of the vertebral artery, and brachial plexus injury. Arteriovenous fistula of the vertebral artery as a complication of direct vertebral arteriography has been reported previously in only four patients (OLSON, HILLIER & SVEN 1963, PHILIPSSON & KARNELL 1956, and SUTTON 1962).

The present report concerns five patients in whom vertebral arteriovenous fistula was the result of percutaneous puncture of the vertebral artery.



Fig 1 Case 1 a) Right vertebral angiography. Perfect placement of the needle with no evidence of extravasation b) and c) Retrograde right brachial angiography. Enlarged proximal right vertebral artery, early filling of vertebral venous plexus (arrows)

Case reports

Case 1 A 33-year-old Negro woman was first seen at the hospital in 1952. Hypertension had been discovered after a transient left-sided hemiparesis when she was 26 years old. In 1960 she complained of sudden frontal headache and vomiting. Minimal residual signs of left-sided weakness were present on neurological examination. The cerebrospinal fluid was grossly bloody.

Bilateral carotid angiography in July 1960 showed no definite abnormality. Attempts at percutaneous puncture of both vertebral arteries were unsuccessful. Percutaneous right vertebral angiography repeated in August 1960 showed no abnormality (Fig 1a). Shortly afterwards the patient noted pain along the distribution of the median nerve in both arms associated with slight hypesthesia. These symptoms gradually subsided.

In December 1960 continuous to-and fro murmurs were heard over both sides of the neck. She was admitted in September 1961 for investigation of a bruit and a machinery-like murmur limited to the right side of the neck. A right common carotid angiography was normal. On Sept 19 1961 retrograde right brachial angiography showed enlargement of the proximal portion of the right vertebral artery and early filling of the vertebral venous plexus (Fig 1b and c). The distal right vertebral and basilar arteries filled well. The left vertebral artery



Fig 2 Case 1 *Left* Transaxillary left vertebral angiography. Enlarged proximal left vertebral artery. No contrast medium in the distal left vertebral artery. Immediate filling of the paravertebral venous plexus. *Right* Later phase. Dilated vertebral vein draining the paravertebral venous plexus.



Fig 3 Case 2 Inadvertent injection into the left vertebral artery. Partial extravasation into the venous plexus.

was not examined at this time. The right vertebral arteriovenous fistula appeared to be at the level of C 5. It was considered asymptomatic and surgical repair was deemed inadvisable.

The patient returned in September 1963 because of sudden onset of slurred speech and right hemiparesis both of which cleared slowly. She had also noted dizzy spells and episodes of generalized weakness. On physical examination a thrill in the left side of the neck below the level of the carotid bifurcation could be felt. A machinery like murmur was noted in the region of both supraclavicular fossae but was more evident on the left side. Transaxillary left vertebral angiography showed a large proximal left vertebral artery patent to the level of C 5 (Fig 2 left view). No contrast material was seen in the distal left vertebral artery. The contrast medium rapidly filled a large paravertebral venous plexus which drained into a dilated vertebral vein (Fig 2 right). Transaxillary right subclavian angiography showed that the right vertebral artery was normal and that the previously noted fistula on the right side had closed spontaneously. No significant relationship appeared to exist between the patient's symptoms and the arteriovenous fistula of the left vertebral artery. Surgical intervention was therefore not advised.

Comment Bilateral vertebral arteriovenous fistulas were the result of attempted puncture of the vertebral arteries. Of interest in this patient is the documented spontaneous closure of the fistula on the right side.



Fig 4 Case 2 a) Left vertebral angiography. Dilated proximal left vertebral artery with immediate filling of the paravertebral venous plexus b) Later phase. Contrast medium with a dilated paravertebral venous plexus right

Case 2 A 38-year-old white woman was first admitted in 1956 for treatment of a long-standing convulsive disorder. Injury to her head at the age of 12 years had been followed by persistent intermittent suboccipital and bi-temporal headaches occasionally associated with transitory vertigo. At the age of 24 she had undergone a right parieto-occipital craniotomy with removal of a chronic subdural hematoma. After the surgical procedure a left hemiparesis had developed and gradually cleared. A year after the operation the first convulsion had occurred. A left craniotomy had been done in 1954 to divide the tentorium.

On physical examination at admission in 1956 a dense symmetrical congruous homonymous quadrantanopia was noted. Bilateral common carotid angiographies showed no abnormalities. During the initial injection on the left however partial filling of the vertebral artery and adjacent venous plexus was observed (Fig. 3).

The patient was readmitted in 1969 complaining of occasional loss of equilibrium and sudden falls without loss of consciousness. On physical examination at that time a loud systolic murmur and a bruit which did not disappear on carotid compression were heard in the left side of the neck. A left vertebral angiography using the femoral artery approach showed that the proximal left vertebral artery was large although its distal caliber appeared normal (Fig. 4a). The contrast medium entered the vertebral venous plexus at the level of C5. On later roentgenograms the contrast medium was seen to cross the midline to fill the vein.



Fig. 2 Case 1. *Left* Transcatheter left vertebral angiography. Dilated proximal left vertebral artery. No contrast medium in the distal left vertebral artery. Immediate filling of the paravertebral venous plexus. *Right* Later phase. Dilated vertebral vein draining the paravertebral venous plexus.

Fig. 3 Case 2. Inadvertent injection into the left vertebral artery. Partial extravasation into the venous plexus.

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The patient returned in September 1963 because of sudden onset of slurred speech and right hemiparesis both of which cleared slowly. She had also noted dizzy spells and episodes of generalized weakness. On physical examination a thrill in the left side of the neck below the level of the carotid bifurcation could be felt. A machinery like murmur was noted in the region of both supraclavicular fossae but was more evident on the left side. Transcatheter left vertebral angiography showed a large proximal left vertebral artery patent to the level of C 5 (Fig. 2, left view). No contrast material was seen in the distal left vertebral artery. The contrast medium rapidly filled a large paravertebral venous plexus which drained into a dilated vertebral vein (Fig. 2, right). Transcatheter right subclavian angiography showed that the right vertebral artery was normal and that the previously noted fistula on the right side had closed spontaneously. No significant relationship appeared to exist between the patient's symptoms and the arteriovenous fistula of the left vertebral artery. Surgical intervention was therefore not advised.

Comment. Bilateral vertebral arteriovenous fistulas were the result of attempted puncture of the vertebral arteries. Of interest in this patient is the documented spontaneous closure of the fistula on the right side.



Fig 6 Case 3 a) Left subclavian angiography. Occlusion of proximal left vertebral artery b) and c) Later phases. Contrast filling of the distal left vertebral artery by collateral channels. Early filling of the vertebral venous plexus and of a dilated vertebral vein (two arrows)

ear synchronous with the heart beat. A loud bruit heard over the entire left neck was not obliterated by compression of the carotid artery. A retrograde aortography suggested the presence of an arteriovenous fistula of the vertebral artery. The bruit heard at the level of C5-6 disappeared after surgical ligation of the proximal left vertebral artery.

Readmitted in October 1966. On physical examination a loud systolic bruit on the left side of the neck was again heard. A left subclavian angiography in January 1963 showed that the proximal portion of the left vertebral artery was occluded (Fig 6a). The ascending cervical artery as well as the branches of the thyrocervical and costocervical trunks communicated with the vertebral venous plexus. The distal left vertebral artery was small and was filled by collateral channels (Fig 6b). Early filling of a prominent vertebral vein was seen (Fig 6c). A right subclavian angiography showed that the fistula was also fed by reversal of flow down the left vertebral artery. Small branches of the right thyrocervical trunk contributed further to flow through the fistula (Fig 7a and b).

Comment. The vertebral arteriovenous fistula resulting from needle trauma was not cured by surgical occlusion of the proximal left vertebral artery. One year after operation the left sided fistula was fed by branches of the left costocervical and thyrocervical trunk. Collateral branches of the right thyrocervical trunk and reversal of flow down the distal left vertebral artery also contributed to the fistula.



Fig. 3. Case 3. Left vertebral angiography. Faulty placement of the needle with immediate filling of vertebral venous plexus with contrast medium.

on the right as well as on the left side (Fig. 4 b). (The puncture of the vertebral artery in 1956 6 years previously had been at the same level as the present arteriovenous fistula.) A right vertebral angiography appeared to be normal with no evidence of reflux into the left vertebral artery. On surgical exploration of the vertebral arteriovenous fistula, arterial blood was seen to be coursing through the vertebral venous plexus adjacent to the vertebral artery. A 5 mm segment of the vertebral artery was resected at the level of the fistula and the thrill and bruit disappeared.

The patient was last seen 1 1/2 years after the operation. At that time the convulsions were controlled medically and no bruits or murmurs were heard in the neck.

Comment. Trauma of the vertebral artery at the time of carotid angiography resulted in vertebral arteriovenous fistula. The fistula was completely obliterated by resection of the vertebral artery at the site of the fistula.

Case 3. When first seen in 1951, a 16 year old white girl had a tremor of the right arm and hand of 2 months duration. On physical examination a rhythmic tremor without increase in tone was present in the right upper extremity. The tremor tended to disappear or decrease with sleep. Bilateral carotid angiographies were normal.

The patient was readmitted in July 1961 complaining of a strange feeling in the right arm as well as headaches of 5 months duration. The tremor in the right arm had continued and the right side was slightly weak. An encephalography as well as left vertebral and left carotid angiographies were normal at that time. The placement of the needle for the vertebral angiography, however, was faulty and some filling of the vertebral veins was noted (Fig. 5).

On readmission in December 1961 the patient complained of a blowing noise in the left

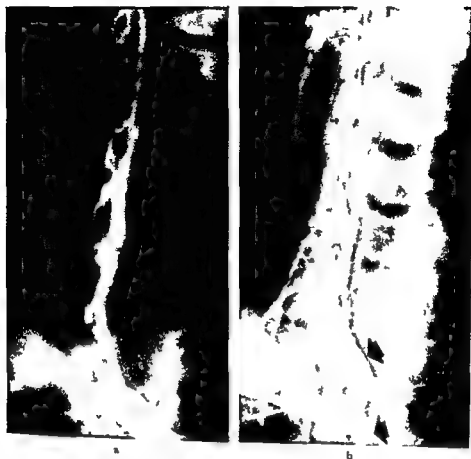


Fig 8 Case 4 a) Transarterial right vertebral angiography. Immediate filling of vertebral venous plexus. Distal vertebral artery is smaller than the proximal vertebral artery. b) Late arterial phase. Early filling of dilated vertebral vein.

the vertebral artery (Fig 8 b). After exploration of the vertebral arteriovenous fistula and ligation of numerous veins in the area, the bruit disappeared.

Comment: A direct vertebral artery puncture resulted in a vertebral arteriovenous fistula.

Case 5: A history of two transient episodes of hemiparesis, each affecting a different side of the body, was obtained from this 36-year-old white man. In July 1960, bilateral carotid angiographies were normal. A right subclavian angiography had failed to demonstrate the vertebral artery. A satisfactory left vertebral angiography had been obtained by direct percutaneous puncture at the level of C5-6. Cerebral vascular occlusive disease had been diagnosed and the hemiparesis had been attributed to transient ischemic attacks.

The patient was first seen at the University of Minnesota Hospital in January 1961, com-



Fig. 7. Case 3. a) Branches of the right thyrocervical trunk contribute to left vertebral arteriovenous fistula. b) Late arterial phase. Reversal of flow down the left vertebral artery represents further contribution to the left vertebral arteriovenous fistula.

Case 4. A 39 year old white man had a sudden episode of severe occipital headache in January 1963. Deep tendon reflexes were slightly hyperactive and slight nuchal rigidity was present. A lumbar puncture revealed blood in the spinal fluid. Bilateral carotid angiographies showed no abnormality. Attempted left vertebral angiography was unsuccessful. The patient was readmitted two months later complaining of occasional occipital headaches. On physical examination no abnormalities were noted. Bilateral carotid angiographies were again normal. A direct right percutaneous vertebral angiography resulted in a partial subintimal and extraluminal injection. At the level of C 2 the vertebral artery was narrow with contrast material extending only to the level of C 1.

The patient was readmitted one year later because of a pulsatile noise in his head. On physical examination a murmur was heard in the right side of the neck. Carotid or jugular compression did not decrease the bruit but rotation of the head to either direction markedly diminished it. At a right vertebral angiography using the transaxillary catheter technique the distal right vertebral artery was relatively small. A fistulous communication between the vertebral artery and venous plexus was observed at the level of C 5 (Fig. 8 a). Later roentgenograms of the same series showed a large single vertebral vein adjacent to the first portion of

Discussion

Previous reports of vertebral arteriovenous fistulas have stressed their rarity. The vertebral artery is well protected within the transverse cervical canal and is therefore not susceptible to injury. Most of the fistulas that have been reported were the result of penetrating injuries to the vertebral artery from either gunshot or stab wounds (CHOU & FRENCH 1965, ELKIN & HARRIS 1946, HEIFETZ 1945, JEFFERSON, BAILEY & KERR 1956, KUTTNER 1917, SHUMACKER 1946). Three patients with vertebral arteriovenous fistulas resulting from blunt trauma to the head or neck (ARONSON 1961, ELKIN and HARRIS 1946, FAETH & DLEKER 1961) and one patient with a spontaneous fistula have been described (GOODY & SCHECHTER 1960). SUGAR, HOLDEN & POWELL (1949) first suggested the possibility of producing a vertebral arteriovenous fistula by damaging the vertebral artery and vein during vertebral angiography or by inadvertently puncturing the vertebral artery at the time of attempted carotid angiography, but only four fistulas have been reported.

Anatomic considerations This paucity of reports of fistulas following puncture of the vertebral artery is surprising considering the anatomic relationship of the vertebral artery to the adjacent venous plexus. This relationship which has been described previously (GREITZ, LILLIEQUIST & MULLER 1962, PERRIG 1931) was confirmed by us by anatomic dissection of these vessels. By injecting colored latex into one of the cervical vertebral bodies the vertebral venous plexus could easily be filled and the dissection simplified. Anatomic roentgenologic studies with injection of contrast medium into a cervical vertebral body also demonstrated the vertebral venous plexus.

The extracranial vertebral artery may be divided into 3 parts. From its origin at the subclavian artery, the first portion extends to its entrance into the transverse foramen usually at the level of the sixth cervical vertebra. The second portion of the artery lies within the canal transversarium and ends at its exit from the atlas. The third portion, suboccipital in location, extends from its exit from the atlas to its entrance into the skull.

In its second portion within the bony canal a venous plexus completely surrounds the vertebral artery. The veins communicate freely with the epidural longitudinal vertebral sinuses, the basivertebral vein and the prevertebral veins (GREITZ, LILLIEQUIST & MULLER 1962). The vertebral venous plexus drains into a single vertebral vein that lies somewhat lateral and anterior to the first portion of the vertebral artery. This vertebral vein enters the internal jugular vein near its junction with the subclavian vein.

Clinical features The diagnosis of a vertebral arteriovenous fistula may be



Fig 9 Case 5 a) Left vertebral angiography. Immediate filling of dilated venous plexus. b) Post operative angiography. Vertebral artery is normal with no evidence of fistula. (From CIRIO & FRENCH 1963. Reprinted with permission.)

plaining of a rhythmic pulsatile noise in the neck, present since the previous angiography. On physical examination a loud bruit with systolic and diastolic components was noted in the neck. The bruit was obliterated by head turning to the right, hyperextension or compression of the carotid artery. Bilateral carotid angiographies showed no abnormality. A right subclavian angiography showed partial arteriosclerotic narrowing at the origin of the right vertebral artery. An arteriovenous fistula of the vertebral artery at the level of C5-6 (Fig 9 a) was revealed on left subclavian angiography. In March 1961 the fistula was explored. Numerous venous channels were noted anterior, medial and lateral to the vertebral artery. These were dissected free of the artery and coagulated. The fistula itself was not identified but the bruit disappeared. A left vertebral angiography in January 1963 showed this vessel to be normal with no evidence of residual fistula (Fig 9 b).

Comment. In this case the fistula apparently followed a direct percutaneous vertebral angiography that was technically uncomplicated. Unlike those in the other cases, this bruit was obliterated by carotid compression. (The data in this case have been reported by CIRIO & FRENCH and have been reprinted with the permission of the authors and of J. Neurosurg.)

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suspected by the presence of a pulsatile bruit in the neck, which is often audible to the patient. In most cases, the murmur is not affected by carotid compression. This clinical test however is not reliable since compression of the carotid artery usually results in simultaneous compression of the jugular vein. The elevation of venous pressure on compression of the jugular vein may decrease the flow through the vertebral arteriovenous fistula and thus stop the bruit.

Accurate angiography, then, is essential to establish the correct diagnosis as well as to determine the level of the fistula. Moreover, demonstration of any collateral arterial supply to the fistula is important in the surgical management of these patients. Branches of the thyrocervical or costocervical trunk may contribute to the fistula, or the flow in the distal vertebral artery may be reversed towards the fistula. Such alternate collateral branches are probably more common in the chronically established fistula, especially one treated by simple ligation of the proximal vertebral artery.

Treatment of vertebral arteriovenous fistulas has been described previously (CHOU & FRENCH 1965, KUTYPER 1917, PERRIE 1931) and is outside the scope of this report. Important to emphasize however is the usual inadequacy of simple ligation of the proximal vertebral artery and the necessity of obliterating the fistula itself.

Radiologic considerations Percutaneous puncture of the vertebral artery probably always results in minor damage to the surrounding venous plexus. Moreover, with imperfect puncture and needle placement, contrast material may occasionally be injected partly into the artery and partly into the venous plexus. In some instances such trauma probably results in temporary arteriovenous communications that close spontaneously.

Arteriovenous fistula of the vertebral artery is easily diagnosed after the injection of contrast medium into the proximal vertebral artery. The exact site of the fistula may be difficult to ascertain however because of overlying contrast filled veins. As with fistulas in other locations, the proximal artery and draining veins were enlarged in most of our patients.

Six other patients, in whom partial venous filling had been noted at vertebral angiography, were re-examined clinically following the angiography. None of these patients had noted any pulsatile noises nor were bruits heard in the neck. Repeated vertebral angiography in 2 of these patients showed the vertebral artery to be intact.

In order to prevent the formation of an arteriovenous fistula following vertebral angiography, direct puncture of the vertebral artery must be avoided. All methods of angiography have certain risks and each investigator must choose an approach which in his hands has the least complications. Since

1963 catheter techniques have been used in vertebral angiography at the University of California Hospital in San Francisco in most instances. In children and young adults the transfemoral route is chosen and a polyethylene catheter is advanced into the left vertebral artery. In older patients the trans axillary approach is used and the catheter is placed into the proximal right vertebral artery. Usually catheterization of the right vertebral artery presents no difficulty but occasionally the catheter cannot be maneuvered easily into this artery. In these instances an injection with the catheter tip in the subclavian artery usually results in adequate contrast filling of the vertebrobasilar vessels. Catheter techniques have advantages other than the prevention of arteriovenous fistula: only local anesthesia with minimal premedication is necessary; a careful unhurried examination in any desired position is also possible; something that is often difficult with direct puncture of the vertebral artery.

SUMMARY

Five patients with angiographically demonstrated vertebral arteriovenous fistulas resulting from percutaneous puncture of the vertebral artery are reported. The intimate relationship of the vertebral artery and its surrounding venous plexus is stressed. The use of catheter techniques is recommended to avoid this complication.

ZUSAMMENFASSUNG

Es wird über 5 Patienten mit angiographisch darstellbaren vertebralen arteriovenösen Fisteln nach perkutaner Punktion der Aorta vertebralis berichtet. Die intime Beziehung der Vertebralarterie mit dem sie umgebenden Venenplexus wird hervorgehoben. Um diese Komplikation zu verhindern wird die Anwendung der Kathetertechnik empfohlen.

RÉSUMÉ

Présentation de cinq malades atteints de fistule artérioveneuse vertébrale prouvée par angiographie et résultant de ponction percutanée de l'artère vertébrale. Les auteurs soulignent les rapports étroits de l'artère vertébrale avec les plexus veineux qui l'entourent. Ils recommandent le cathétérisme pour éviter cette complication.

REFERENCES

- ARONSON N. J. Traumatic arteriovenous fistula of the vertebral vessels: angiographic demonstration and a rationale for treatment. *Neurology* 11 (1961) 817.
- CAIRO S. N. and FRENCH L. A. Arteriovenous fistula of vertebral vessels in the neck. *J Neurosurg* 24 (1965) 77.
- FLINN D. C. and HARRIS M. H. Arteriovenous aneurysm of vertebral vessels: report of 10 cases. *Ann Surg* 124 (1946) 934.

- FAETH, W. H. and DUINKER, H. W. Arteriovenous vascular malformation of the cervical portion of the vertebral artery: a case report. *Neurology* 11 (1961) 492.
- GOODY, W. and SCHIECHTER, M. M. Spontaneous arteriovenous fistula of the vertebral artery: case report. *Brit J Radiol* 33 (1960) 709.
- GREITZ, T., LUNDFQUIST, B. and MÜLLER, R. Cervical vertebral phlebography. *Acta radiol* 57 (1962) 353.
- HEIFETZ, C. J. Traumatic aneurysm of first portion of left vertebral artery: case report. *Ann Surg* 122 (1945) 102.
- JEFFERSON, C., BAILEY, R. A. and KERR, A. S. Suboccipital arteriovenous aneurysms of vertebral artery. *J Bone Jt Surg* 38B (1956) 114.
- KUTTNER, H. Die Verletzungen und traumatischen Aneurysmen der Vertebralgefäße am Halse und ihre operative Behandlung. *Beitr z klin Chir* 108 (1917) 1.
- LINDGREN, F. Percutaneous angiography of vertebral artery. *Acta radiol* 33 (1950) 589.
- MONES, R. Vertebral angiography, an analysis of 106 cases. *Radiology* 76 (1961) 750.
- OLSON, R. W., HILLIER, L. B. JR and SIEN, H. J. Arteriovenous fistula: a complication of vertebral angiography, report of a case. *J Neurosurg* 20 (1963) 73.
- PERRIC, H. Zur Anatomie, Klinik und Therapie der Verletzungen und Aneurysmen der Arteria vertebralis. *Beitr z klin Chir* 154 (1931) 272.
- PHILIPSON, J. and KARNFLI, J. Arteriovenous fistel efter punktion av a vertebralis for cerebral angiografi. *Opusc med (Stockh)* 1 (1956) 49.
- SHUMACKER, H. B. JR. Arteriovenous fistulas of cervical portion of vertebral vessels. *Surg Gynec Obstet* 83 (1946) 625.
- SILVERMAN, E. M., BERGMAN, P. S. and BENDER, M. B. The dynamics of transient cerebral blindness: report of nine episodes following vertebral angiography. *Arch Neurol* 4 (1961) 333.
- SUGAR, O., HOLDEN, L. B. and POWELL, C. B. Vertebral angiography. *Amer J Roent enol* 61 (1949) 166.
- SUTTON, D. *Arteriography*. E & S Livingstone Ltd, Edinburgh, 1962.

LE PHLEBOGRAMME ORBITAIRE

par

G OFFRET D ARON ROSA J METZGER et D DOYON

L'exploration du système veineux de l'orbite a déjà fait l'objet de plusieurs travaux en France notamment ceux de BODER (1953—1954)

Si l'on connaît bien grâce à l'anatomie classique aux ouvrages spécialisés (WOLFF 1961 HEPPY 1959) l'aspect anatomique du réseau veineux orbital on ne peut en dire autant de son aspect radiologique

Avec le concours de Professeur Fischgold nous avons essayé de codifier l'interprétation de la phlebographie orbitaire

Technique Après dénudation de la veine angulaire sous anesthésie locale nous cathétérisons la veine directement avec une aiguille à injection intra-veineuse de calibre moyen reliée à une seringue contenant du sérum physiologique par un tube de 10 cm de long en plastique (polyéthylène). Une fois l'aiguille bien en place le sang reflue à l'aspiration et la seringue se vidant sous faible pression du piston on installe le malade pour l'incidence de profil, ensuite de face faisant couler du sérum constamment entre chaque injection de produit opaque pour éviter la coagulation dans l'aiguille. 7 ml de Vasurix 25 ou de Radioselectan à 60 % sont injectés à la seringue automatique sous 3 kg de pression ou à défaut à la main en 2 ou 3 secondes

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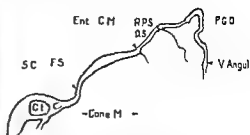


Fig 1 Hélobogramme orbitaire normal de profil
I C O indique poulie du grand oblique R
P S releveur de la paupière supérieure D S
droit supérieur Ent C M entrée dans le cône
musculaire F S passage dans la fente sphé-
noïdale S C sinus caverneux C I carotide
interne V Angul veine angulaire

films de 1 par seconde, un premier film est pris au temps 0 juste avant l'injection pour servir à la soustraction, le dernier film est tiré à la 15^{ème} seconde. On se contente d'un cliché toutes les deux ou trois secondes à partir de la 3^{ème} seconde, nous utilisons généralement 7 films pour chaque incidence.

Le générateur et le tube de rayons roentgen doivent pouvoir donner des temps de pose courts, de l'ordre de 1/10 de seconde.

Tous les clichés sont soustraits pour obtenir un meilleur contraste de l'image par effacement des ombres osseuses nombreuses au niveau de l'orbite.

Les incidences

1 De profil, cliché centré sur l'orbite, en diaphragmant au maximum pour améliorer les contrastes, avec des constantes légèrement inférieures à celles d'un profil standard du crâne,

2 De face occiput plaque, rayon passant par le milieu de l'orbite incliné d'environ 10° sur le plan de Virchow dans le sens crudo crânial, pour que les rochers se projettent au dessous de la clarté orbitaire, en diaphragmant, mais en radiographiant simultanément les deux orbites.

3 Incidence axiale. Nous n'avons fait que quelques essais de cette incidence difficile à réaliser, l'aiguille quittant la veine lors de la mobilisation de la tête. Cette incidence s'annonce pourtant intéressante.

Plus récemment nous avons commencé à mesurer systématiquement les pressions veineuses (par un manomètre de Claude gradué en centimètres d'eau) au repos, après manœuvre de compression des jugulaires et après arrêt de cette compression.

Tolérance de l'examen. L'injection du produit opaque est indolore.

La cicatrice secondaire et la dénudation est minime passant aussi inaperçue que celle d'une incision pour intervention sur le sac lacrymal.



Fig 2 Phlebogramme orbitaire normal de profil autre variété opacification de la veine ophtalmique inferieure V A indique veine angulaire V O I veine orbitaire inferieure

Nous n'avons eu aucun accident et seulement trois incidents sur une trentaine d'examen : une injection sous cutanée qui s'est resorbée sans douleur en quelques minutes et deux veines angulaires non cathérisables. Dans un de ces cas une veine frontale a été injectée par erreur la veine angulaire n'ayant pu être repérée. Dans l'autre cas il s'agissait d'une malade opérée à qui l'on avait lié la veine ophtalmique supérieure.

Enfin il faut signaler que pour l'ophtalmologue et le radiologue, il n'y a pas de risque d'irradiation avec le sénographe et la seringue automatique, la prise des clichés et la seringue étant déclenchés d'une cabine de commande protégée.

Resultats

Phlebographie normale

Les images des courants veineux de l'orbite et de la veine ophtalmique supérieure en particulier sont suffisamment constantes pour qu'on puisse en faire un schéma morphologique topographique et chronologique (hémodynamique) auquel pourront être comparées les diverses images pathologiques rencontrées.

Divers points devront être étudiés : la morphologie des images veineuses et leur place dans l'orbite sur les clichés de profil et de face ; le remplissage éventuel du système anastomotique et du système veineux orbitaire ; la latéralité ; l'opacification du sinus caverneux et ses différents types ; enfin nous décrivons les premiers résultats de l'étude des pressions dans la veine angulaire et de la mesure des temps de circulation veineuse du système orbito-caverneux.

Phlebogramme orbitaire en incidence de profil La veine ophtalmique supérieure ou principale (Fig 1) sinueuse fait suite à l'angulaire et pénètre dans l'orbite

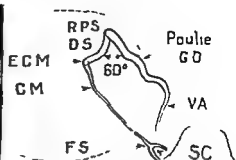


Fig. 3 Phlebogramme orbitaire normal de face. Angle supérieur de 60° à l'entrée de la veine dans la gaine du droit supérieur (D S) et du releveur de la paupière supérieure.

(R P S) E C M indique le point d'entrée dans le cône musculaire. Remarquer la longueur du trajet libre dans le cône musculaire (C M). V A indique veine angulaire. F S fente sphénoïdale. S C sinus caverneux.

bridée par la poulie du grand oblique, qui lui fait décrire une anse concave en bas, c'est le premier point fixe au delà duquel elle reçoit souvent une veine accessoire, elle parcourt ainsi le 1/3 supérieur de l'orbite.

La veine chemine, plaquée contre le toit de l'orbite au dessus du cône musculaire, au dessus donc des releveurs et droit supérieur.

À l'union du 1/3 antérieur et du 1/3 moyen de l'orbite, la veine ophtalmique supérieure s'incline par un mouvement oblique en bas et en arrière et sur un court trajet contourne le bord latéral des releveurs et du droit supérieur, plaquée contre leur gaine, pénétrant ensuite dans le cône musculaire. C'est le 2ème point fixe.

À l'intérieur du cône la veine se dirige en arrière et en bas, croise dans l'espace le nerf optique et sort par la fente sphénoïdale pour aboutir au pôle antérieur du sinus caverneux.

Dans son trajet elle reçoit (Fig. 2) des veines musculaires et deux veines vertigineuses ainsi que les collatérales, rarement et incomplètement visibles.

Quelquefois on trouve individualisée une grele veine inférieure, réunie à la veine ophtalmique supérieure par une ou plusieurs anastomoses verticales, c'est la veine ophtalmique inférieure, simple veine musculaire qui suit le plancher de l'orbite et se jette, soit dans la partie terminale de la veine ophtalmique supérieure, soit directement dans le sinus caverneux.

Quant à la veine ophtalmique moyenne, située entre ces deux veines et encore plus grele nous ne l'avons jamais individualisée.

Le calibre de ces veines est assez variable. Radiologiquement, la limite maximum normale de la veine ophtalmique supérieure est de 1,5 à 2 mm, celle de la veine ophtalmique inférieure de 1 à 1,5 mm.

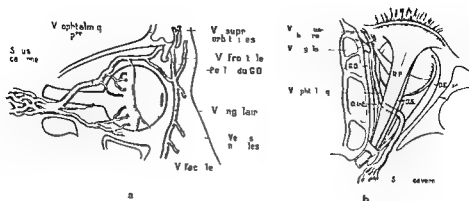


Fig 4 Schémas anatomiques des veines de l'orbite a) Vue externe b) Vue supérieure G O grand oblique R P releveur de la paupière D S droit supérieur D E droit externe D Int droit interne

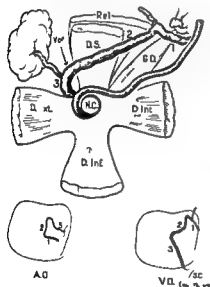


Fig 5 Schéma anatomique et projection radiologique des veine et artère ophtalmique Vue de face On peut décrire trois segments 1 — de la poulie du grand oblique (G O) à la gaine des muscles droit supérieur (D S) et releveur (Rel) 2 — segment dans la gaine des muscles droit supérieur et releveur 3 — segment intra-conique N O nerf optique D ext droit externe D int droit interne D inf droit inférieur

L'opacification bilatérale des systèmes veineux orbitaires rend la lecture des images difficile sur le profil

En conclusion de profil existent d'importants repères fixes dans ce phlébogramme poulie du grand oblique plafond de l'orbite pénétration dans le cône musculaire arrivée au sinus caveux ce sont les segments contenus entre les points fixes qui modifient leur aspect sous l'influence des déplacements tumoraux

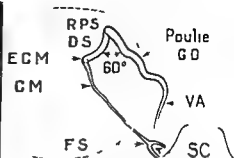
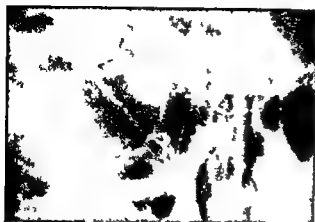


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Quelquesfois on trouve individualisée une grêle veine inférieure, réunies à la veine ophtalmique supérieure par une ou plusieurs anastomoses verticales, c'est la veine ophtalmique inférieure, simple veine musculaire qui suit le plancher de l'orbite et se jette, soit dans la partie terminale de la veine ophtalmique supérieure, soit directement dans le sinus caverneux.

Quant à la veine ophtalmique moyenne, située entre ces deux veines et encore plus grêle, nous ne l'avons jamais individualisée.

Le calibre de ces veines est assez variable radiologiquement, la limite maximum normale de la veine ophtalmique supérieure est de 1,5 à 2 mm, celle de la veine ophtalmique inférieure de 1 à 1,5 mm.

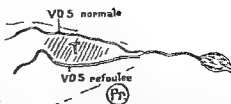


Fig 7 Cas 1 (Voir aussi Fig 6) Phlebogramme orbitaire de profil. Les deux veines orbitales supérieures (VOS) étant opacifiées on voit l'importance du refoulement en bas par la tumeur (T) de la veine orbitale supérieure du côté atteint. A noter de nombreux anastomoses.

L'opacification du système veineux orbitaire controlatéral semble pathologique les systèmes anastomotiques souscutanés frontaux et temporaux étant généralement peu visibles. La veine faciale est presque toujours bien opacifiée ainsi que le plexus ptérygoidien réuni à celle-ci et au sinus caverneux, l'opacification du système veineux controlatéral fournit un repère comparatif excellent dans l'appréciation du côté lésionnel.

L'aspect radiologique du sinus caverneux Décrit par de nombreux auteurs il est variable. Il se projette sur le versant latéral de la selle turcique et prend souvent un aspect de resille vasculaire semblant perforé au centre par le contour rond de la carotide interne en particulier sur les clichés de face. Parfois il n'est formé que par deux ou trois veines à trajet antéro-postérieur sur le cliché de profil mal individualisables sur le cliché de face.

Enfin rarement il n'est formé que d'une grosse veine de plusieurs millimètres de diamètre, type de description classique des anatomistes. Pour le situer sur le cliché de face il suffit de repérer les pointes des rochers il se projette juste en dedans à leur hauteur.

En cas d'opacification des deux systèmes veineux orbitaires les deux sinus caverneux sont généralement visibles.

Chronologie des images veineuses lors de l'injection. A la première seconde la partie antérieure du système veineux est seule opacifiée. A la deuxième et à la troisième seconde le système veineux orbitaire est entièrement opacifié. Le sinus caverneux paraît visible de la 3ème à la 5ème seconde. La persistance du produit opaque au-delà de la 5ème seconde dans le système veineux orbito-caverneux devient suspecte.

Dans les tumeurs orbitaires il est entre les points fixes que se font les dé-

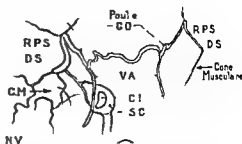


Fig 1 Cas 1 Phlébogramme orbitaire de face injecté bilatéralement avec opacification de la communicante antérieure joignant les deux veines angulaires (V A) A droite l'angle supérieur du phlébogramme est fermé (in est très diminuée il existe des neo-vaisseaux C O grand oblique S C sinus caveux C I carotide interne C M cône musculaire (Tumeur de l'orbite extra conjuguée)

inférieur à 30°) la surface du parallélogramme en dehors (N V) R I S indique releveur de la paupière supérieure D S droit supérieur C O grand oblique S C sinus caveux C I carotide interne C M cône musculaire (Tumeur de l'orbite extra conjuguée)

Phlébogramme orbitaire de face Dans son ensemble, la veine ophtalmique de face (Fig 3), décrit un parallélogramme ouvert en bas, compris dans le 1/3 interne de l'orbite

Un premier segment, oblique en haut et en dehors, continue l'angulaire, fixe à l'angle supéro interne de l'orbite par la poulie du grand oblique, il rejoint le bord antérieur du toit orbitaire à l'union du 1/3 moyen et du 1/3 interne. La veine change alors brusquement de direction, les deux segments formant un angle de 50° à 60°. Le deuxième segment décrit un arc concave en dehors, la veine contourne le releveur et le droit supérieur, appliquée sur l'aponévrose. Une seconde fois, la veine change de direction. Le troisième segment, toujours à l'intérieur du cône est oblique en dedans et en bas, semblant longer le bord supérieur de la fente sphénoïdale pour se jeter dans le sinus caveux sur le versant latéral de la selle turque.

En conclusion on peut schématiser l'image de face comme un parallélogramme à trois cotés supéro interne et inféro externe rectilignes, réunis par un côté supéro externe concave en dehors, formant un angle supérieur aigu d'environ 60° ouvert en bas et un angle inférieur obtus d'environ 110°. En cas de compression tumorale, angles et aspect des cotés se modifient.

Le calibre est là encore inférieur à 2 mm mais il faut signaler que parfois les deux cotés de l'angle supérieur se superposent à son origine faisant croire à une dilatation veineuse, parfois la veine ophtalmique supérieure a un aspect dédoublé du fait d'un canal collatéral.

Les autres branches veineuses sont rarement individualisées chez le sujet normal.

males et dans la partie interne et inférieure de l'orbite un croissant veineux anormal très volumineux (5 mm). De profil (Fig 9 b) sur la partie initiale de la veine ophtalmique supérieure deux petites dilatations anévrysmales mais la malformation majeure siège à la partie postérieure de l'orbite elle a la forme d'un tronc de cône moulé par le cone musculaire. Son sommet répond juste à celui de l'orbite.

La station du produit opaque dans la malformation nous a gêné pour le tirage du cliché négatif de soustraction la malformation reste injectée jusqu'à la 13ème seconde.

La pression veineuse de départ est de 15 cm pour monter à 27 après compression des jugulaires et descendre à 16 après décompression.

Une autre anomalie (hors de l'orbite) est à signaler c'est une petite malformation veineuse mise en évidence dans le sinus maxillaire.

RÉSUMÉ

La phlébographie semble un complément indispensable de l'artériographie dans l'exploration vasculaire de l'orbite. En cas de tumeur elle permet de préciser le siège extra ou intracônique de la tumeur aidant le choix de la voie d'abord. Surtout sur les clichés de face en cas de malformation vasculaire elle a permis d'expliquer la récurrence des saignements intraorbitaux après résection des portions antérieures des angiomes. Elle a permis de voir que épaisses ou en résille fine ces malformations avaient toujours un pôle antérieur et un pôle postérieur (expliqués par l'embryologie). Ceci soulève un problème de voie d'abord postérieure de la malformation très difficile non seulement à cause du siège mais du groupement à ce niveau de tout le paquet vasculo-nerveux de l'orbite.

SUMMARY

Phlebography would seem to be an indispensable complement to arteriography in vascular exploration of the orbit. In tumor cases it gives good information regarding the extra or intracônical site of the growth and assists the choice of the way of approach. Especially in frontal views in cases of vascular malformation it helped to explain recurrences of intraorbital bleeding after resection of the anterior portions of the angiomas. It was possible to establish that these malformations whether thick or thin always had anterior and posterior poles (explained by embryology). This creates a difficult problem with respect to the posterior approach to the growth not only because of the site but also because of the concentration of the whole bundle of vasculo-nervous structures of the orbit at this level.

ZUSAMMENFASSUNG

Für die Gefäßuntersuchung der Orbita scheint die Phlebographie eine unerlässliche Ergänzung zur Arteriographie zu sein. Bei Tumoren erhält man gute Information über die extra- oder Intrakonische Ausbreitung die die Wahl des Operationsweges erleichtert. Besonders durch Frontalprojektionen von Gefäßmissbildungen trug die Untersuchung zu einer Erklärung von Reziden von intraorbitalen Blutungen nach Resektion der vorderen Teile der Angiome bei. Es war möglich klarzustellen dass diese Missbildungen ob sie nun dick oder haardünn sind immer einen vorderen und hinteren Pol haben was embryologisch erklärt werden kann. Es ergibt sich somit ein schwieriges Problem im Hinblick auf den Zugang zum hinteren Pol nicht nur wegen der Lage sondern auch wegen der dort befindlichen Konzentration von Nerven und Gefäßen.

En conclusion le diagnostic de tumeur maligne orbitaire sus-conique fut posé et confirmé par l'histologie qui révéla un réticulo-sarcome.

Mais l'aspect de phlébogramme est différent au cours des tumeurs situées à l'intérieur du cône musculaire dont voici un exemple.

Cas 2. Neurinome orbito-frontal avec envahissement du sinus caverneux. Femme de 19 ans présente un syndrome de la fente sphénoïdale avec exophtalmie, atrophie optique, atteinte des III^e, IV^e, V^e et VI^eème nerfs. Sur les clichés standard on note la destruction de la fente sphénoïdale et une opacité du sinus maxillaire droit. Sur l'arteriographie, une vaste tumeur fronto-temporale déplaçant la cérébrale antérieure et la cérébrale moyenne, peu ou peu de déplacement de l'artère ophtalmique.

La phlébographie évocatrice surtout sur l'image de face (Fig. 8) permet d'affirmer l'existence d'une tumeur dans l'orbite de la situer et de découvrir l'envahissement du sinus caverneux: on notait de face une ouverture des angles veineux avec élargissement du phlébogramme déformé et parcouru par d'importants néovaisseaux. Cet aspect évoque le siège intra-conique de la tumeur.

Par ailleurs la constatation d'une pression angulaire basse (3 cm d'eau) non modifiée par la compression jugulaire, le rétrécissement de la veine ophtalmique supérieure dans sa partie terminale avec l'absence d'injection du sinus caverneux de face et de profil, l'injection massive du système ophtalmique opposé et des collatérales sous-cutanées superficielles permettent de diagnostiquer l'envahissement certain du sinus caverneux.

Toutes choses confirmées par l'examen de la pièce anatomique et l'examen histologique de ce neurinome malin envahissant le sinus caverneux.

Les malformations vasculaires veineuses

Elles restent l'une des meilleures indications de la phlébographie orbitaire.

En matière de malformation vasculaire, l'arteriographie carotidienne, même par la carotide externe est souvent normale (10 fois sur 13 malades explorés). Le reste du temps, la malformation est veineuse et seule la phlébographie permet de la retrouver.

Dans l'ensemble, ces malformations veineuses sont caractérisées: 1) cliniquement par des hémorragies récurrentes dans l'orbite, intra-vitréennes ou sous-conjonctivales avec parfois angiome nœudiforme conjonctival ou palpébral, 2) radiologiquement par une hyperpression veineuse avec énorme veine ophtalmique supérieure et paquet angiomatueux à deux poles. L'un très antérieur dans l'orbite et l'autre postérieur devant la fente sphénoïdale, enfin le ralentissement de la circulation dans la malformation, l'anomalie restant injectée au delà de la sixième seconde.

Cas 3. Hématomes rétro-orbitaires et intra-vitréens gauches récurrents. Homme de 12 ans. Arteriographie carotidienne gauche normale.

Phlébographie. Le calibre de la veine ophtalmique supérieure est quadruplé (7 mm) et les affluents de l'ophtalmique supérieure sont extrêmement visibles. De face (Fig. 9a) il existe sur la racine supérieure de la veine ophtalmique deux petites malformations nœudiformes anévris-

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DIAGNOSTIC VALUE AND COMPLICATIONS OF CAROTID AND VERTEBRAL ANGIOGRAPHY

by

G PERRET

The Cooperative Study of Intracranial Aneurysms and Subarachnoid Hemorrhage sponsored by the United States Public Health Service through the National Institute of Neurological Diseases and Blindness, represents the combined efforts of 19 university neurologic and neurosurgical centers in the United States and one in England. It was established to study clinical and therapeutic aspects of subarachnoid hemorrhage and aneurysms. The following centers participate in this study: Baylor University (Houston), University of Buffalo, University of California (San Francisco), Columbia University and New York Neurological Institute, Cornell University and Bellevue Hospital (New York), Duke University, Harvard University and Massachusetts General Hospital, University of Indiana, University of Iowa, University of London and Atkinson Morley's Hospital, University of Louisville, University of Michigan, University of Minnesota, Mount Sinai Hospital in New York, University of Pennsylvania, University of Tennessee, Washington University, St. Louis, Wayne State University (Detroit), University of Georgia.

As of June 1, 1964, the Central Registry at the University of Iowa had received 2,828 cases which included 2,946 cases with intracranial aneurysms (50.3%).

REFERENCES

- BOUDIER CH. Premiers résultats de la phléboangiographie orbitaire. Thèse Montpellier 1953—1954
 — La place du phléboangiogramme orbitaire dans le diagnostic des tumeurs de l'orbite. Arch. Ophthal. 15 (1955) 597.
- BRECIAT P., DAVID M. et FISCHCOIN M. Exploration du sinus caverneux par phléboangiographie orbitaire. Bull. Soc. Ophthal. 33 (1952) 336.
- DEJEAN CH., VIALEFONT O., BOLDEFI CH. et PAVCHA O. Neurofibromatose de Recklinghausen à localisation orbitaire. Presse méd. 61 (1953) 80.
- HENRY J. Contribution à l'étude de l'anatomie des vaisseaux de l'orbite et de la loge cavernueuse (par injection de matières plastiques) du tendon de Zinn et de la capsule de Tenon. Thèse Paris 1959.
- KRAMERBUH H. The value of orbital angiography. J. Neurosurg. 19 (1967) 289.
- OFFRET G., ARON ROSA D. et DUFRENE D. Intérêt de l'opacification des vaisseaux dans les angiomes orbito-palpébraux. Arch. Ophthal. 23 (1963) 275.
- WOLFE S. F. Anatomy of the eye and orbit. Lewis, London 1961.

angiographies were performed with negative results 14 died and one internal carotid aneurysm was identified at autopsy If there is any suspicion that an aneurysm may be present even though the initial carotid angiograms are negative repeat angiography should be carried out for confirmation of the diagnosis especially if the angiography was done within the first four days after hemorrhage Three or four vessel angiography should routinely be carried out

Complications of angiography can be divided into local, systemic and neurologic The local complications arise at the site of vessel puncture and are directly connected with the technique of puncture and injection of contrast material The systemic complications may reflect toxicity of the contrast material and sensitivity of the patient to the contrast material The more serious complications are those arising from the central nervous system and are represented by new abnormal neurologic signs or symptoms or by aggravation of previously existing abnormal neurologic signs or symptoms An analysis of 5 669 angiographies showed that there was a total of 333 studies with immediate or delayed complications This represents an incidence of 6 0% 4 3% were immediate and 1 75% were delayed However among these complications are included 31 cases of recurrent subarachnoid hemorrhage which occurred at the time of or shortly after the angiography It is very doubtful that the subsequent subarachnoid hemorrhage in a patient who has had such hemorrhages before and has an aneurysm is a true complication of angiography It may represent the natural course of the initial disease In our studies there were 363 carotid angiographic studies performed in patients who had aneurysms but who never bled In none of these cases was carotid angiography followed by a subarachnoid hemorrhage Thus if the subsequent subarachnoid hemorrhage was discarded the incidence of true angiographic complications is 5 5% The complications included transient hemiparesis in 1 4% of the cases permanent hemiparesis in 0 5% convulsions in 0 5% and severe neck hematoma in 0 5%

A study of complications in various age groups showed that there were twice as many complications after the age of 60 as before the age of 20 but only in patients who have ruptured aneurysms Age had little influence on the complications of angiography in the presence of subarachnoid hemorrhage not caused by ruptured aneurysm The nature of the contrast material had little influence on the complications except for thorotrast which produced six complications in 30 cases Thorotrast may have been used in patients in poor condition or known to be allergic to other media

Analysis of the incidence and nature of angiographic complications in relation to the number of injections or to the amount of contrast material

452 cases of arteriovenous anomalies (7.7 %), and 58 cases with both aneurysm and arteriovenous anomaly (0.99 %). There were 2,402 cases of subarachnoid hemorrhage not due to aneurysm or arteriovenous anomalies (41 %) and 521 cases of non bleeding aneurysm or arteriovenous anomaly. The site of single aneurysm was on the internal carotid artery in 44.3 %, anterior communicating region in 29.1 %, middle cerebral artery in 19 %, pericallosal artery 2.3 % and posterior portion of the circle of Willis in 5.3 % of cases. The incidence of multiple aneurysms as disclosed by angiography was 15 %, while in a series of 644 autopsies, multiple aneurysms were found in 23 % of cases. Sixteen per cent had two aneurysms, 4.2 % three aneurysms and 2.6 % four or more aneurysms.

The angiographic studies were carried out under local anesthesia in 78 % of cases and under general anesthesia in 22 %. Hypaque was the contrast material used in 90 % of the cases. The number of injections varied greatly between one and nine, and the volume of injected contrast material varied between one and 101 ml.

The question has frequently been raised as to whether there is any relationship between the demonstration of the aneurysm and the time interval between the subarachnoid hemorrhage and angiography. It has been postulated that vascular spasm and other alterations in cerebral circulation immediately after subarachnoid hemorrhage may result in an aneurysm not becoming filled with contrast medium if angiography is performed too soon. In our study, bilateral carotid angiography was carried out within the first seven days after hemorrhage in 66 % of the cases and within two weeks after hemorrhage in 80 % of the cases. On the basis of 4,364 angiographies carried out at various intervals after the bleeding episode from one day to over 30 days, an aneurysm could be demonstrated in 45.5 %. The incidence did not vary more than ± 4.5 %, suggesting that the time of angiography in relation to hemorrhage has no significant effect on the chances of demonstrating aneurysm if one is present.

In 993 cases which failed to reveal an aneurysm or arteriovenous malformation after subarachnoid hemorrhage on initial bilateral carotid angiography, further carotid angiography was performed in 158 cases and revealed, in 35 cases, an aneurysm of the carotid system presumably undetected in the first examinations. This represents a 3.5 % false negative report on initial bilateral carotid angiography. A comparison of this figure with autopsy findings is of interest. One hundred and five patients with negative bilateral carotid angiographic findings died and post mortem examination revealed the presence of an aneurysm in 26. 12 on the anterior communicating, 3 on the middle cerebral, 2 on the anterior cerebral, 2 on the internal carotid artery and 7 on the vertebral basilar circulation. Of a series of cases in whom two carotid

angiographies were performed with negative results 14 died and one internal carotid aneurysm was identified at autopsy If there is any suspicion that an aneurysm may be present even though the initial carotid angiograms are negative repeat angiography should be carried out for confirmation of the diagnosis especially if the angiography was done within the first four days after hemorrhage Three or four vessel angiography should routinely be carried out

Complications of angiography can be divided into local systemic and neurologic The local complications arise at the site of vessel puncture and are directly connected with the technique of puncture and injection of contrast material The systemic complications may reflect toxicity of the contrast material and sensitivity of the patient to the contrast material The more serious complications are those arising from the central nervous system and are represented by new abnormal neurologic signs or symptoms or by aggravation of previously existing abnormal neurologic signs or symptoms An analysis of 5669 angiographies showed that there was a total of 333 studies with immediate or delayed complications This represents an incidence of 6.0%, 4.3% were immediate and 1.75% were delayed However among these complications are included 31 cases of recurrent subarachnoid hemorrhage which occurred at the time of or shortly after the angiography It is very doubtful that the subsequent subarachnoid hemorrhage in a patient who has had such hemorrhages before and has an aneurysm is a true complication of angiography It may represent the natural course of the initial disease In our studies there were 363 carotid angiographic studies performed in patients who had aneurysms but who never bled In none of these cases was carotid angiography followed by a subarachnoid hemorrhage Thus if the subsequent subarachnoid hemorrhage was discarded the incidence of true angiographic complications is 5.5% The complications included transient hemiparesis in 1.4% of the cases permanent hemiparesis in 0.5%, convulsions in 0.5% and neck hematoma in 0.5%

A study of complications in various age groups showed that there were twice as many complications after the age of 60 as before the age of 20 but only in patients who have ruptured aneurysms Age had little influence on the complications of angiography in the presence of subarachnoid hemorrhage not caused by ruptured aneurysm The nature of the contrast material had little influence on the complications except for thorotrast which produced six complications in 30 cases Thorotrast may have been used in patients in poor condition or known to be allergic to other media

Analysis of the incidence and nature of angiographic complications in relation to the number of injections or to the amount of contrast material

injected showed no direct relationship with either factor. The incidence of complications seemed to be higher in the patients who were examined under general anesthesia as compared to those examined under local anesthesia. Under general anesthesia, the rate of immediate and delayed complications was increased and so was the rate of permanent hemiparesis. Interestingly enough, the rate of convulsions was the same (0.5 %). It seems that the complication rate from angiography is not influenced by the patient's state of responsiveness at the time of the study. However, it was interesting to note that the incidence of convulsion was lower in the alert patient than in the patient with disturbances of responsiveness. The patient's pre angiographic neurologic status did not seem to influence the rate of complications.

The incidence of complications is greater in the presence of subarachnoid hemorrhage. Of the patients examined angiographically after their first bleeding episode, those with neither aneurysm nor arteriovenous anomalies had a lower incidence of complication than those who had an aneurysm. Patients who had an aneurysm or arteriovenous anomaly but who had not had a subarachnoid hemorrhage showed an even lower complication rate. Most patients having a ruptured aneurysm had a higher complication rate during the first two weeks following hemorrhage than when the study was done at a later date. This may suggest that angiography may add an additional insult to an already impaired cerebral circulation.

Complications from postoperative angiography were higher in patients who were treated with craniotomy than in those treated by an intracranial operation, 5.1 % and 2.2 % respectively. This may again be directly related to the disturbance in cerebral circulation and the increased impairment of circulation produced by angiography.

The incidence of fatal angiographic complications is difficult to assess. Arbitrarily, it was considered that any death occurring within 24 hours of angiography or due to permanent complications should be included. In a group of 3,972 patients who were studied by angiography, 39 died during or after angiography, representing a gross mortality rate of 0.98 %. If, however, one excludes rebleeding from angiographic complications, the mortality rate directly related to the angiographic procedure was 0.6 %. Thirteen patients had a proven recurrent hemorrhage and died: six bled during the procedure, and seven bled after completion of the procedure. Seven patients who were moribund prior to angiography died during or shortly after the examination. Three patients became suddenly unresponsive during the procedure and died 1, 2 and 18 days later without regaining consciousness and without recurrent hemorrhage. Four patients developed a hemiplegia during angiography and died within 24 hours. Two patients became hemiplegic after completion of the

procedure, one died within 24 hours and was found to have a thrombotic occlusion of the basilar artery after vertebral angiography had been done. The other died six days later. Three patients developed a cervical hematoma requiring tracheostomy and died within 24 hours. One of these patients had a gross hematoma tracking down into the mediastinum. One patient had a convulsion during the angiography and died three days later. One patient became hypotensive and died suddenly three hours later. Three patients progressively deteriorated dying within five days, one was shown at autopsy to have a dissecting aneurysm of the common carotid artery. Two patients who had angiography performed under general anesthesia were hemiplegic and aphasic on awakening and both died within three days.

Conclusion A total of 5 685 angiographic studies performed upon 3 972 patients have been analyzed. The incidence of aneurysms missed in the usual bilateral carotid angiograms points out the need for repeated examination (at angiography) including the vertebral basilar system. Study of the complications does not allow us to make any specific recommendations as no factors have been proven to be significantly related to the complications.

The final report based on 7 932 angiographies is published in the *Journal of Neurosurgery* July 1966.

SUMMARY

A report is presented on 2 946 cases with intracranial aneurysms or vascular anomalies with special emphasis on statistical distribution, angiographic demonstrability and complications risks.

ZUSAMMENFASSUNG

Es wird über 2 946 Fälle mit intrakraniellen Aneurysmen oder Gefässmissbildungen berichtet wobei der statistischen Verbreitung, angiographischen Darstellbarkeit und den Komplikationsrisiken besondere Beachtung geschenkt wird.

RÉSUMÉ

Présentation de 2 946 cas d'anévrismes intracrâniens ou anomalies vasculaires avec étude particulière de leur distribution statistique, de la possibilité de les mettre en évidence par angiographie et des risques de complications.

injected showed no direct relationship with either factor. The incidence of complications seemed to be higher in the patients who were examined under general anesthesia as compared to those examined under local anesthesia. Under general anesthesia, the rate of immediate and delayed complications was increased and so was the rate of permanent hemiparesis. Interestingly enough, the rate of convulsions was the same (0.5%). It seems that the complication rate from angiography is not influenced by the patient's state of responsiveness at the time of the study. However, it was interesting to note that the incidence of convulsion was lower in the alert patient than in the patient with disturbances of responsiveness. The patient's pre-angiographic neurologic status did not seem to influence the rate of complications.

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Présentation de 2,946 cas d'anévrismes intracrâniens ou anomalies vasculaires avec étude particulière de leur répartition statistique, de la possibilité de les mettre en évidence par angiographie et des risques de complications.

TRANSAXILLARY SELECTIVE CATHETERIZATION OF THE CAROTID AND VERTEBRAL ARTERIES

by

PLINIO ROSSI

The axillary approach to arterial catheterization is a recent advent in angiography. With the advance of medical techniques and improved specialized equipment such as catheters, guide wires, pressure injectors, and image intensification, angiography has become increasingly important as a diagnostic aid.

Most techniques used to inject contrast medium into the aortic arch and its branches with a single injection result in a suboptimal demonstration of the intracerebral vessels. This is due to the excessive dilution of the contrast medium within the aorta and brachiocephalic trunk. In addition, simultaneous contrast filling of all the vessels of the head results in superimposition of the arteries with loss of valuable information.

To overcome these shortcomings, we have used a technique whereby, through a single puncture of the axillary artery, the major vessels of the neck may be demonstrated and then selectively catheterized for completion of the study of the intracerebral circulation (Figs 2, 3, 4 and 5). The entire examination is performed without additional punctures of the other arteries leading to the head.

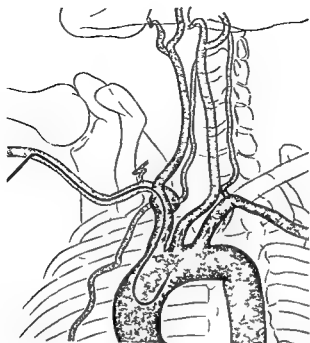


Fig 1 Transaxillary catheterization of the aortic arch. Arrows indicate the direction in which the catheter may be advanced for selective contrast injection into the major vessels in the neck.

This angiographic technique is based upon the use of a specially shaped catheter similar to one described by AMPLATZ et coll (1963) and a modified Seldinger wire.

The catheter is made of radio opaque polyethylene approximately 50 cm in length with a U shaped tapered tip. This catheter is shaped by taking a conventional red kifa, inserting a guide wire and heating the tip over an alcohol flame. Gradual tension results in a narrowing of the lumen of the catheter so that the inner diameter of the heated section becomes that of the guide wire. Once the tip is formed the excess is cut off and the catheter with the wire still within its lumen is immersed in very hot water and the tip bent into a loop. It is then plunged into cold water so that the present shape will be permanent. One additional hole is made at the side of the catheter 1 cm from the end hole to allow easy flow of contrast medium and avoid recoil.

The Seldinger guide wire is modified by partial removal of its core so that its flexible tip is increased to about 6 inches in length. This modified guide wire we have named floppy wire because of its long flexible end.

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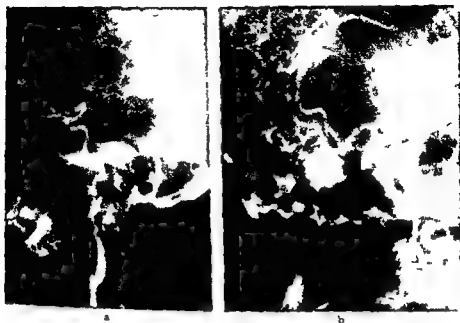


Fig 4 Lateral project on (a) and oblique project on (b) Arterial phase Good demonstration of the intracerebral vessels

undesired arteries. If no obstruction is met the wire is advanced into the ascending aorta and the U shaped catheter then slipped over the wire which is subsequently removed. If the wire cannot be advanced due to tortuosity, arteriosclerotic plaques or abnormal position of the mouth of the vertebral or other arteries the floppy wire is then used.

The use of a long flexible tip has proven to be a considerable aid in traversing these obstacles. This can be done because the tip of the floppy wire becomes anchored at the outer wall of a bend in the artery and the flexible portion loops on itself and follows the lumen of the tortuous artery acting as a new tip which has already passed the site of temporary obstruction without applying any direct pressure on the obstacle.

When the catheter is finally placed within the aorta and the wire withdrawn the catheter tip will return to its preformed U shape (see Fig 1). As can be seen from the figure the tip of the catheter is now pointing at the orifice of the major arteries arising from the aortic arch and by proper manipulation the tip can be made to enter the selected artery. This is accomplished by slow withdrawal of the catheter trying to hook the tip in the mouth of the artery. If technical difficulties prevent proper placement of the catheter the floppy



a



b



a



b

Fig 2 a) The flexible portion of the wire has entered the right common carotid artery. Position of wire indicates the artery entered. b) Following removal of the wire a test injection is made to control the position of the tip of the catheter.

Fig 3 a) The flexible portion of the wire has entered the left common carotid artery. b) Following removal of the wire and contrast injection the artery is clearly demonstrated.

Method

The patient is sedated with 100 mg of Pentobarbital sodium (Seconal)[®] given one hour before the procedure and 50 mg of Meperidine hydrochloride (Demerol)[®] and atropine sulphate 0.4 mg just prior to transfer to the roentgen department.

For the examination, the patient is placed on the radiographic table in a minimal degree of right posterior oblique position with the selected arm abducted 90 degrees and externally rotated. The elbow is flexed and the hand supported by a foam rubber pad. This position is used to open the aortic arch so as to identify the anatomic structures and tip of the catheter under fluoroscopy. Although this technique has been used with both conventional fluoroscopy and image intensification, it is found that in heavy patients identification of the position of the tip of the catheter is very difficult without image intensification. The right axillary approach is usually selected as it facilitates ease of the examination.

Careful palpation of the axilla is made to determine the site of maximum pulsation and course of the axillary artery. The skin is then entered up proximately 2 cm from the outer margin of the great pectoralis muscle.

After puncturing the artery with the needle a Seldinger wire is advanced into the artery and its passage observed under fluoroscopy to avoid entering

4 Using this remote portal of entry, it is safe to place the catheter in the mouth of a major vessel when marked narrowing or occlusion of the other vessel is present

5 The floppy wire has less chance of displacing an atheromatous plaque

6 Multiple arterial punctures of the neck are obviated when panangiography is required

7 Superimposition of vessels is avoided since only a single artery is filled with contrast medium at one time

8 Excellent filling of the intracranial vessels is obtained (Fig. 4)

9 Rate of filling of both sides of the head may be compared since the contrast enters from the arch of the aorta

10 The arteries of the neck are not compromised since the vessels are not punctured

11 Radial pulse is usually preserved during and after the procedure

12 There is much less discomfort to the patient

13 A long and tortuous femoral artery course is obviated

Results

Thirty five cases have been examined using this technique. In 65 % of the cases successful results were obtained. A successful result is defined as contrast filling of the aortic arch and major arteries of the neck and of the intracerebral vessels. On occasion spasm of the vessels has prevented a successful completion of the study.

In two cases, the left vertebral artery arose from the aortic arch and in one case the left common carotid artery was occluded.

In one case the radial pulse was absent for 2 days and petechial hemorrhages appeared on the arm.

SUMMARY

This paper presents a study of selective catheterization of the vessels leading to the head using a single arterial puncture the axillary artery. Techniques are described for shaping catheters and guide wire to facilitate the selective catheterization. Results and advantages are discussed.

ZUSAMMENFASSUNG

Es wird über eine Studie von selektiver Katheterisierung der Gefäße des Kopfes mittels einfacher Punktion der Art. axillaris berichtet. Es wird die Technik der Formung des Katheters und Führungsdrahtes zwecks Erleichterung der selektiven Katheterisierung beschrieben. Ergebnisse und Vorteile der Methode werden besprochen.



Fig. 5 Lateral projection of the vertebral artery arterial phase. The catheter was advanced from left axillary artery into left vertebral artery for several centimeters.

wire is then reintroduced until its tip extends beyond the catheter end. Due to the flexibility of the wire the catheter shape is not disturbed and the tip of the wire will point in the same direction as the catheter.

By combining manipulation of the catheter and wire, the tip of the wire can usually be placed in the desired position. The position of the tip indicates the artery entered (Figs 2 and 3). Once the wire is in place, the catheter can then be introduced by following the leading wire.

The wire is then withdrawn and 5 ml of contrast medium injected through the catheter to verify its position (Figs 2 and 3). If placement is correct, 12 ml of contrast medium are injected by hand or by pressure injection at low pressure to demonstrate the intracranial portion of the artery and the study is carried out in the usual fashion (Figs 4 and 5).

Advantages of the method

- 1 Using a single puncture, the arch of the aorta and its major branches may be filled with contrast medium.
- 2 Without removing the catheter, selective contrast filling of each of the major vessels leading to the head may be obtained.
- 3 Tortuous vessels may be negotiated using the 'floppy wire'.

4 Using this remote portal of entry it is safe to place the catheter in the mouth of a major vessel when marked narrowing or occlusion of the other vessel is present

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Fig. 5. Lateral projection of the vertebral artery, arterial phase. The catheter was advanced from left axillary artery into left vertebral artery for several centimeters.

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CONSIDERATIONS RADIOLOGIQUES DANS LES NEURINOMES DU GANGLION DE GASSER

par

R. RUBERTI F. GALLIGIONI et A. CARTERI

Les neurinomes du ganglion de GASSER sont des tumeurs rares, tout au moins si on compare leur fréquence à celle des tumeurs du nerf acoustique. Depuis les descriptions de plusieurs auteurs leur symptomatologie est bien connue. Il s'agit de tumeurs à évolution très lente qui se traduisent pendant longtemps par des algies faciales.

Toutefois la seule clinique est très souvent insuffisante pour le diagnostic de ces tumeurs tandis que l'étude radiologique peut nous aider considérablement à résoudre ce problème.

L'étude radiologique de ces malades a été fondée dans le passé surtout sur l'examen standard et sur la pneumographie on n'a pas suffisamment souligné par contre, à notre avis l'importance que l'angiographie carotidienne et vertébrale peuvent avoir dans le diagnostic de ces tumeurs.

C'est pour cela que nous croyons utile de faire quelques considérations radiologiques basées soit sur les données de la littérature soit sur 6 cas de neurinome du ganglion du GASSER contrôlés chirurgicalement à la Clinique Neuro-Chirurgicale de Padoue. Dans nos 6 cas il s'agissait de tumeurs à siège exclusivement ou surtout extra-dural 2 strictement limitées à la fosse cérébrale

RÉSUMÉ

Présentation d'une étude du cathétérisme sélectif des artères céphaliques par une seule ponction artérielle de l'artère axillaire. Description de la technique pour donner aux cathéters et au fil guide la forme qui facilite le cathétérisme sélectif et discussion des résultats et des avantages.

REFERENCES

- AMPLATZ K and HARNER R A new subclavian catheterization technic *Radiology* 78 (1962) 693
- REICH J and HILSI S A catheter approach for cerebral angiography *Radiology* 81 (1963) 675
- BAUM S and ABRAMS H I A J shaped catheter for retrograde catheterization of tortuous vessels *Radiology* 83 (1964) 430
- BERG D VAN DEN et coll Vertebrobasilar angiography via catheterization of the brachial artery *Ned T Geneesk* 107 (1963) 174
- COIFFIER J et coll Vertebral arteriography by axillary route Preliminary results in vascular syndromes of the brain stem *Ann Radiol* 7 (1964) 189
- GOREFF J A TINDALL G T and ODOM J L Percutaneous retrograde brachial angiography in the diagnosis of acoustic neurinoma — Results in 4 cases *Amer J Roentgenol* 97 (1964) 829
- HANALIF W Axillary artery approach to carotid vertebral abdominal aorta and coronary angiography *Radiology* 81 (1963) 559
- HAUT I Catheter vertebral angiography *Acta radiol Suppl* No 109 (1964)
- HOWLAND W J and CURRY J L Transient cerebral blindness — A hazard of vertebral artery catheterization *Radiology* 83 (1964) 428
- KLAMUT M CZOCHRA M BRYL S and ZDRZEWIECZ F A technique for right vertebral arteriography with the aid of carotid catheterization *Neurol Neurochir Psychiat pol* 17 (1962) 699
- MARSHALL T R LING J F and GONZALES P Additional experience with direct percutaneous noncatheter bronchial angiography — left panarteriography — right cerebral angiography *Radiology* 81 (1963) 568
- MULLER R GREITZ I LILJQVIST B and HELLSTROM L Aorticercival angiography in occlusive cerebrovascular disease *Neurology* 14 (1964) 136
- NEWTON T R The axillary artery approach to arteriography of the aorta and its branches *Amer J Roentgenol* 89 (1963) 275
- PYGOTT F and HUTTON C F Vertebral arteriography by percutaneous brachial artery catheterization *Brit J Radiol* 32 (1959) 114
- RADNER S Vertebral angiography by catheterization New method employed in 221 cases *Acta radiol Suppl* No 87 (1951)
- RIVERA R and PEDROTE J A Bilateral vertebral angiography by one contrast injection *J cardiovasc Surg* 4 (1963) 101
- ROY P Percutaneous catheterization of the axillary artery New route of approach for carotid and vertebral arteriography *Roentgen Europ* 6 (1963) 122
- SCOTT M HURTAGH I LAPAYOWKER M and BAIRD M Vertebral basilar and carotid angiography by injection of brachial artery *Amer J Roentgenol* 90 (1963) 546



Fig 3 Artériographie vertébrale droite dans un autre cas de neurinome du ganglion de Gasser droit à développement sus- et sous-tentorial comme le montre pour ce qui concerne l'expansion sous-tentoriale le déplacement considérable de l'artère basilaire et de ses branches

moyenne 4 a développement predominant dans la fosse cérébrale moyenne mais avec une expansion sous tentorielle

En ce qui concerne l'examen radiographique standard le signe le plus typique que nous avons observé dans tous nos cas et dans plusieurs cas de la littérature est sûrement constitué par la lyse à contours nets du bord supérieur du rocher au niveau de la fossette du ganglion de GASSER lyse qui peut arriver suivant l'expansion de la tumeur jusqu'à la destruction de toute la partie interne du rocher. L'ostéolyse de ces structures donne lieu naturellement à la typique image de l'élargissement du trou déchiré antérieur par érosion des bords interne et externe du trou lui-même. Tout à fait exceptionnels sont l'élargissement des trous ovale et grand rond la lyse des clinoides postérieures et des autres structures qu'on peut en réalité trouver dans les neurinomes très volumineux, mais qui n'ont aucun caractère d'utilité diagnostique. Les données de l'examen standard peuvent pourtant fournir une première orientation dans le diagnostic.

Pour ce qui concerne l'examen pneumographique il permet d'avoir des renseignements précis sur l'expansion de la tumeur mais ne donne aucune information utile au point de vue de sa nature car il permet très difficilement d'établir le siège intra- ou extra-dural de la lésion.

C'est incontestablement l'arteriographie carotidienne qui constitue de loin l'examen le plus instructif car ses données sont à la fois riches et précises.

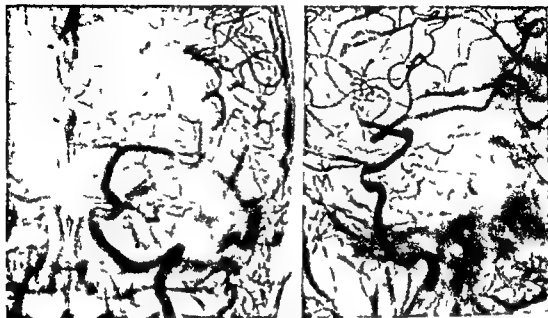


Fig. 1 Artériographie carotidienne gauche. Déformation du segment extra dural de la carotide interne particulièrement évident dans la projection antéro postérieure



Fig. 2 Figures schématiques reproduisant la déformation du segment extra dural de la carotide interne dans les neurinomes du ganglion de Gasser. Le déplacement est caractéristique et permet le diagnostic du siège et de la nature de la lésion

etc.) et veineux indiquent seulement l'expansion réelle de la tumeur dans la fosse cérébrale moyenne

Dans les tumeurs qui ont une expansion sous tentorielle l'angiographie vertébrale peut fournir des renseignements précis. En effet le déplacement éventuel du segment supérieur de l'artère basilaire est pathognomonique de l'expansion sous tentorielle de la tumeur (Fig 3) tandis que le soulèvement de l'artère cérébrale postérieure ne l'indique pas avec certitude (Fig 4)

En conclusion le diagnostic de neurinome du ganglion de Gasser peut souvent être affirmé radiologiquement. L'atteinte caractéristique du rocher et le déplacement du segment extra dural de la carotide interne ont une valeur diagnostique précieuse

RÉSUMÉ

Cette étude radiologique est basée sur 6 cas de neurinome du ganglion de Gasser vérifiés chirurgicalement. Les auteurs soulignent en particulier l'utilité de l'artériographie carotidienne pour le diagnostic de l'expansion extra durale de la tumeur et de l'artériographie vertébrale pour le diagnostic de son extension sous tentorielle

SUMMARY

Some radiological aspects of six cases of surgically verified neurinomas of the gasserian ganglion have been studied. The importance of carotid angiography in regard to the extradural extension of these lesions and of vertebral angiography in regard to the subtentorial extension is stressed

ZUSAMMENFASSUNG

Einige röntgenologische Gesichtspunkte von sechs chirurgisch verifizierten Neurinomen des Ganglion Gasseri werden mitgeteilt. Der Wert der Carotisangiographie bei extraduraler Ausbreitung und die Wichtigkeit der Vertebralangiographie bei subtentorieller Ausbreitung solcher Veränderungen wird hervorgehoben

BIBLIOGRAPHIE

- BERNASCONI V. Caratteristiche radiologiche dei processi espansivi extradurali del pavimento della fossa cranica media. *Radiol med* (Torino) 45 (1959) 725
- KRAYENBUHL H. Das Neurinom des Nervus Trigemini. *Bull. Schweiz. Acad. med. Wiss.* 15 (1959) 89
- LÖNNER E. Das Röntgenbild bei Tumoren des Ganglion Gasseri. *Acta chir. scand.* 85 (1941) 181
- TUNNIS W. und PIA H. W. Die Geschwülste der mittleren Schädelhöhle in Arteriogrammen. *Zbl. Neurochir.* 12 (1959) 145
- RUBERTI R. Considerazioni angiografiche su un caso di tumore parasellare extradurale. *Riv. Anat. pat.* 10 (1955) 196
- WESTBERG G. Angiographic changes in neurinoma of the trigeminal nerve. *Acta radiol. I D* (1963) 513



Fig. 4. Arteriographie vertébrale gauche dans un troisième cas de neurinome du ganglion de Gasser gauche développé exclusivement dans la fosse cérébrale moyenne. Le soulèvement de l'artère cérébrale postérieure est modéré et n'indique pas avec certitude une expansion sous tentorielle de la tumeur.

L'artériographie carotidienne montre l'extension extra durale de la tumeur par le déplacement caractéristique du segment extra durale de la carotide interne (Fig. 1), déplacement qui est complètement différent de ceux provoqués par d'autres tumeurs extra durales de la même région (ostéochondrome, sarcome, kyste épidermoïde etc.). Alors que les autres tumeurs extra durales de la fosse cérébrale moyenne donnent un déplacement en haut et en dedans du segment extra durale de la carotide interne, les neurinomes du ganglion de Gasser déplacent le même segment artériel en bas et en dedans. Ce déplacement artériel caractéristique déterminé par les neurinomes du ganglion de GASSER est mieux compréhensible si l'on considère la situation anatomique particulière du ganglion lui-même et la modalité d'expansion de la tumeur par rapport aux structures ostéo durales et vasculaires contigües. Le siphon carotidien, au niveau du trou déchiré antérieur et dans son trajet extra durale, semble délimiter en dedans et en bas la tumeur située à la pointe du rocher (Fig. 2).

Les déplacements des autres segments artériels (artère cérébrale moyenne,

etc.) et veineux indiquent seulement l'expansion réelle de la tumeur dans la fosse cérébrale moyenne.

Dans les tumeurs qui ont une expansion sous tentorielle l'angiographie vertébrale peut fournir des renseignements précis. En effet le déplacement éventuel du segment supérieur de l'artère basilaire est pathognomonique de l'expansion sous tentorielle de la tumeur (Fig. 3) tandis que le soulèvement de l'artère cérébrale postérieure ne l'indique pas avec certitude (Fig. 4).

En conclusion, le diagnostic de neurinome du ganglion de Gasser peut souvent être affirmé radiologiquement. L'atteinte caractéristique du rocher et le déplacement du segment extra dural de la carotide interne ont une valeur diagnostique précieuse.

RÉSUMÉ

Cette étude radiologique est basée sur 6 cas de neurinome du ganglion de Gasser vérifiés chirurgicalement. Les auteurs soulignent en particulier l'utilité de l'artériographie carotidienne pour le diagnostic de l'expansion extra-durale de la tumeur et de l'artériographie vertébrale pour le diagnostic de son extension sous tentorielle.

SUMMARY

Some radiological aspects of six cases of surgically verified neurinomas of the gasserian ganglion have been studied. The importance of carotid angiography in regard to the extradural extension of these lesions and of vertebral angiography in regard to the subtentorial extension is stressed.

ZUSAMMENFASSUNG

Einige röntgenclogische Gesichtspunkte von sechs chirurgisch verifizierten Neurinomen des Ganglion Gasseri werden mitgeteilt. Der Wert der Carotisangiographie bei extraduraler Ausbreitung und die Wichtigkeit der Vertebralisangiographie bei subtentorieller Ausbreitung solcher Veränderungen wird hervorgehoben.

BIBLIOGRAPHIE

- BERSACCHI V. Caratteristiche radiologiche dei processi espansivi extradurali del pavineto della fossa cranica media. *Radiol. med. (Torino)* 45 (1959) 795.
- KAAVENSTAD H. Das Neurinom des Nervus Trigemini. *Bull. schw. Akad. med. Wiss.* 15 (1959) 89.
- LENDREY E. Das Röntgenbild bei Tumoren des Ganglion Gasseri. *Acta chir. scand.* 85 (1941) 181.
- TOYNE W. und PIA H. W. Die Geschwulste der mittleren Schädelgrube in Arteriogramm. *Zbl. Neurochir.* 12 (1959) 145.
- REBERTI R. Considerazioni angiografiche su un caso di tumore parasellare extradurale. *Riv. Anat. pat.* 10 (1955) 196.
- WEITNER G. Angiographic changes in neuroma of the trigeminal nerve. *Acta radiol. (D)* (1963) 513.

ANATOMIC-RADIOLOGIC STUDY OF THE SMALL SUPERFICIAL VESSELS OF THE BRAIN

by

SANDRO SASSAROLI, TOMMASO DI GIULIO and ANTONIO BACIOCCO

Between the emptying of the arteries and before definite filling of the major veins small vessels roughly parallel and resembling railroad tracks (Fig. 1) are visible on serial angiograms. They are situated on the brain surface and in a lateral film they form a compound ribbon like image whose bends closely follow the brain sulci and border the convolutions.

The deep vessels belonging to the white matter are branches of the surface network, entering the various layers of the cortex as short, middle or long pre capillary vessels. Most of the small arterial branches are arranged in convergent fashion and pass the white matter in the direction of the lateral ventricles. The deepest vessels are the perforating basal arteries, which in general appear to branch concentrically around the basal nuclei. This pattern of distribution is the same in all parts of the brain (KAPLAN 1956). During the angiographic intermediate phase the vessels of the white matter are not individually recognizable on the film. They only contribute to the diffusely increased density of the brain which is normal in this phase. The ventricles may stand out as a faint negative image against the brain substance (Fig. 2).

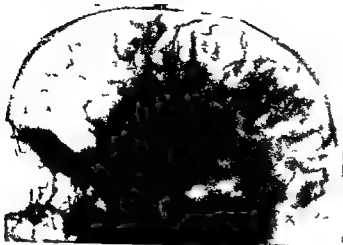


Fig 1 The small vessel is in the precapillary phase giving an impression of the anatomy of the sulci and convolutions

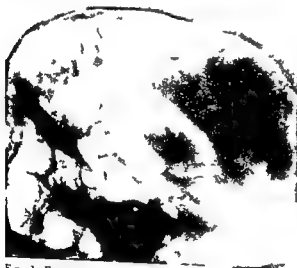


Fig 2 Faint negative image of the lateral ventricle during angiography

A number of the smallest vessels branching on the cortex cross the convolutions at random while others especially little veins emerging from the convolutions lie in the sulci. In a lateral film the vessels of the convexity and of the medial flat surface of the hemisphere overlap. The average diameter of vessels

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Fig 4



Fig 5

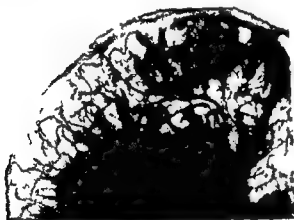


Fig 6

Fig 4 Transverse section of an injected specimen

Fig 5 Frontal pole hollowed out by removal of white matter showing the pattern of the superficial vessels. Contrast material has leaked into the sulci from ruptured vessels in the sulci.

Fig 6 The same appearance in another case before the brain was removed from the skull.

the smaller the vessels concerned in a displacement the more significant is the finding for localization purposes. In fact in the case of tumors the displacement of large arteries or veins is a compromise between the focal pressure and the brain changes due to herniations. This is particularly true of avascular superficial tumors or with a growth which impinges on the cortex (Figs 7, 8 and 9).

Our observations led to the following conclusions:

1. A number of fine superficial vessels, particularly small veins, are crowded into the sulci and consequently allow the observer to gain a general idea of the shape of the cortex at angiography.



Fig. 3 Coronal section through body of lateral ventricles and basal ganglia. The brain was previously injected through the internal carotid. The superficial vessels are clearly recognizable: a number are running in the sulci. Some of the smallest arteries cross at random through the convolutions. Concentric arrangement of white matter vessels which are so small that they are not recognizable as single vessels in an angiogram of the living.

occupying the sulci ranges from 300 to 500 microns, their thickness is difficult to evaluate on the films because some of them lie close together and may overlap, causing a compound image (Fig. 1). The short vessels branching within the cortex may also overlap, thus contributing to the formation of the convolutional angiographic pattern.

In order to study the degree to which the contrast filled vessels of the cortex and of the white matter contribute to the angiographic image during the precapillary phase, we made an anatomical investigation. We injected 15 human brains with variable amounts of vermilion suspension in a colloidal solution. Films were made of these brains or of their sections immediately after the injection or after fixation (Figs 3 and 4). We succeeded in obtaining films similar to those reported by LAZORTHES (1961) and to one reported by SCHÖEN-MACKERS & VIETEN (cf. their Fig. 46). In some cases the images were particularly interesting because there was leakage of the contrast medium into the sulci caused by rupture of some of the smallest vessels (Figs 5 and 6). We found that these small superficial vessels are particularly valuable in giving an idea of the general shape of the cortex. Sometimes we may gain more information from the behavior of these vessels than from larger ones and we feel that as a rule

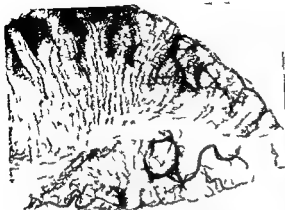


Fig 4

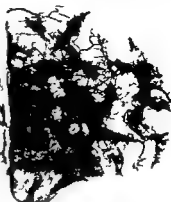


Fig 5

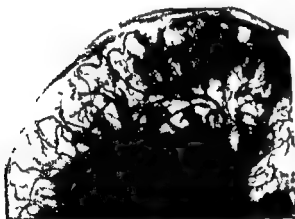


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SUMMARY

Anatomic and radiologic investigations show the possibility of recognizing the convolutions and sulci of the brain in the intermediate phase of carotid angiography. The visible vessels of the cortex during this phase are often particularly useful in the demonstration of expansive lesions.

ZUSAMMENFASSUNG

Anatomische und radiologische Untersuchungen geben die Möglichkeit die Windungen und Furchen des Gehirns in der intermediären Phase einer Carotisangiographie zu erkennen. Die in dieser Phase an der Hirnoberfläche sichtbaren Gefäße sind für die Diagnose von expansiven Processen oft sehr wichtig.

RESUME

Des recherches anatomiques et radiologiques ont démontré la possibilité de reconnaître les convolutions et les sillons du cerveau à la phase intermédiaire de l'angiographie carotidienne. Les vaisseaux corticaux visibles à cette phase sont souvent particulièrement utiles pour le diagnostic de lésions expansives.

REFERENCES

- KAPLAN, H. A. Arteries of the brain. In anatomic study. *Acta radiol.* 46 (1956) 364.
LAZAROTIS, G. Vascularisation et circulation cérébrales. Masson, Paris 1961.
SCHOENMACKERS, J. und VIETEN, H. Atlas postmortalen Angiogramme. Thieme Verlag Stuttgart 1954.

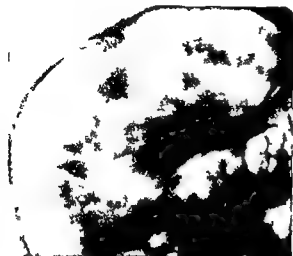


Fig 7 Appearance of the sulci and convolutions in a frontal tumor



Fig 8 Sulci and convolutions in a frontoparietal tumor

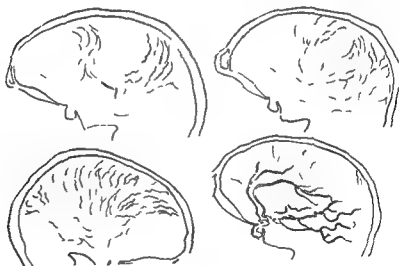


Fig 9 Diagrammatic representation of the displacement of the superficial vessels in different tumor localizations

2 Changes of the width of the sulci or the convolutions (narrowness or enlargement) are the result of pressure or an infiltrating lesion but may sometimes also be found in atrophic conditions

3 The circular arrangement of the sulci surrounding an avascular area is a common localizing sign

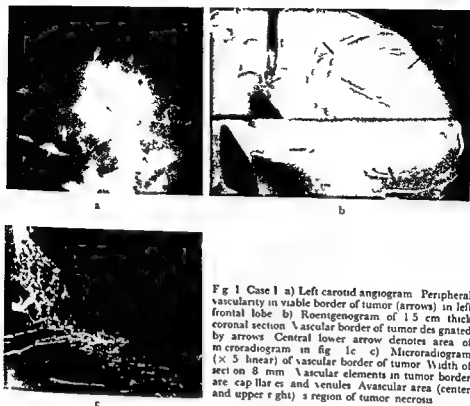


Fig 1 Case 1 a) Left carotid angiogram. Peripheral vascularity in viable border of tumor (arrows) in left frontal lobe. b) Roentgenogram of 1.5 cm thick coronal section. Vascular border of tumor designated by arrows. Central lower arrow denotes area of necrosis. c) Microradiogram ($\times 5$ linear) of vascular border of tumor. Width of section 8 mm. Vascular elements in tumor border are capillaries and venules. Avascular area (center and upper right) is region of tumor necrosis.

Technique

The brain is injected immediately after removal from the cranium. Both hemispheres are injected via the internal carotid and basilar arteries. Open or torn arteries of significant size are ligated prior to injection. The contrast agent (one part Thorotrast, two parts Micropaque, and three to four parts water) is injected slowly by hand. Satisfactory internal brain filling is usually present when fine surface arteries are outlined with contrast material and this is obtained with approximately 30 to 50 ml of contrast agent per hemisphere.

The brain is allowed to fix in the usual fashion (two to three weeks, 20% formal saline). Roentgenograms of the gross brain after sagittal sectioning are then made. These are followed by roentgenograms of the appropriate coronal sections. Photographs of these coronal sections are also made for subsequent correlation.

Using the coronal section films as a guide, microradiography of thick (to 1 cm) and thin (to 3 mm) tumor slices are made. The microradiographic unit

FROM THE DEPARTMENT OF RADIOLOGY, SCHOOL OF MEDICINE, YALE UNIVERSITY
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POST MORTEM BRAIN TUMOR ANGIOGRAPHY

by

JAMES H SCATLIFT, M R CROMPTON and J W D BULL

Although considerable knowledge now exists concerning the *in vivo* angiographic appearance of brain neoplasms little or no radiographic evaluation of tumor vessels in these lesions has been carried out at post mortem. The reasons for this have included unsatisfactory filling of tumor vessels by available contrast agents, surgical alteration of tumours, and limitations in supply of tumor material at any one institution.

In 1961, CHANG & TREMBLEY (2) using a combination of Thorotrast and finely ground barium sulphate (Micropaque) produced excellent filling of renal and cerebral capillaries in several animal species. This mixture of contrast materials has now been used to initiate a survey of the angio architecture of primary brain tumors and cerebral metastases. The material for this study has been derived from a series of fifty brain injections carried out at two institutions (St Georges Hospital London (Atkinson Morley's Unit) and Yale University Medical Center) over an eighteen month period.

The purpose of this report is to describe the injection and radiographic technique employed and present several cases which illustrate the method. Preliminary observations will also be made concerning the tumor vasculature which has been analyzed.

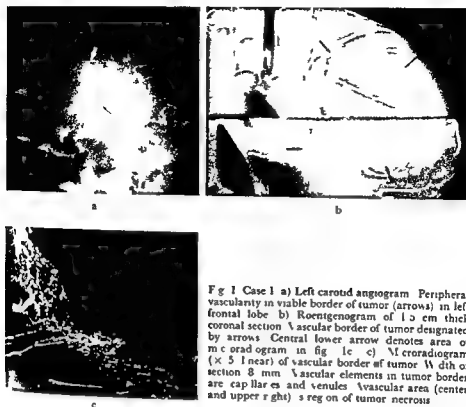


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a



b



c

Fig 3 Case 3 a) Left carotid angiogram late arterial phase. Faint contrast filling of tumor vessels at arrow. The tumor had a cystic component as shown during burr hole biopsy. b) Roentgenogram of 1.5 cm coronal section of solid portion of tumor. Ovoid network of vessels within the tumor (arrow). c) Microangiogram ($\times 15$) of vascular formation as in fig 3 b. Thickness of section 3 mm. Capillaries connected with thick walled venules indicating a well formed arteriovenous shunt.

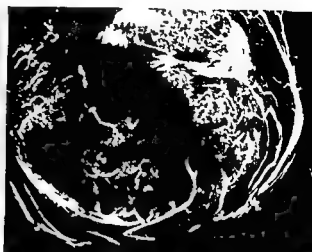
Illustrative cases

Case 1 The patient was a 60 year-old female with progressive right hemiparesis and severe impairment of speech. Left carotid angiography revealed a ring like tumor stain in the left frontal lobe (Fig 1 a). The patient expired one day after burr hole biopsy. A roentgenogram of a coronal section 1.5 cm thick (Fig 1 b) showed the tumor vessels to be well filled with contrast material. A microangiogram of an 8 mm thick section of the lesion (Fig 1 c) outlined a band of capillaries and venules of irregular configuration in the viable tumor border. The histology of the tumor was astrocytoma grade III (1).

Case 2 The 47 year-old male had a three month history of lethargy and personality disturbance. A slowly developing right hemiparesis was also noted. Left carotid angiography produced a rounded well defined tumor stain in the region of the left basal ganglia and thal-

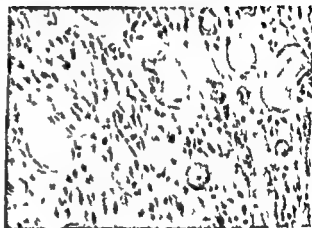


a



b

Fig. 2 Case 2. Roentgenogram of coronal section (1.5 cm) shows well vascularized astrocytoma in thalamus and basal ganglia (arrows). Dilatation of lenticulostriate arteries supplying tumor. Upper arrow denotes area of contrast material extravasation in tumor. b) Microradiogram ($\times 2$) of 8 mm thick section of tumor taken from coronal slice shown in fig. 2 a. Abrupt ending of capillaries in areas of central necrosis (vascular regions in center and lower left of tumor). c) Histologic section ($\times 100$). Capillaries from 10–25 micra in tumor well filled with contrast material.



c

used for the majority of the specimens contains a Machlett type O 2 defraction tube with a 1 mm focal spot $3\frac{1}{4} \times 4$. Kodak projector slide plates have proved satisfactory for filming. Histologic sections (7 micra) are then made from the same slices for tumor classification and evaluation of vessel types and degree of vessel filling.

The microradiograms are then viewed microscopically or may be examined after photographic enlargement. The films of the microradiograms for this report were made by first producing an intermediate negative (Cronar film Dupont) with a photographic enlarger. The intermediate film was then copied with a Log Etronic printer. In this way the microradiographic image can be enhanced as well as enlarged 10 to 20 times with satisfactory preservation of detail.

in the red blood cells and benzidine nitroprusside, Pickworth stain (11) or acid fuchsin EROS (4) will produce a deposition of dark pigment in the vessel lumina. The demonstration of tumor capillaries is dependent on a blood filled vascular bed prior to the application of the dye. Agonal changes in the vessels as well as handling and sectioning of tissues may disperse erythrocytes to such an extent that only a partial outline of tumor vessels is obtained. In addition tissue sections used in staining procedures are generally 150 to 300 micra thick and the resulting patterns seen have a scattered or fragmented appearance.

A second histologic approach of greater merit is the use of silver stains as recommended by COURVILLE (3). Silver impregnation of the reticulin in the vessel wall would appear to give a more thorough outline of vessels at a capillary level and the illustrations of the results of this method are quite striking. The width of the sections of approximately 250 micra poses a limitation however similar to that found in erythrocyte staining methods. It is evident that vascular formations (see Fig. 3c) with dimensions greater than 250 micra may be transected in thin sections and go unrecognized.

Another technique advocated for the study of cerebral vessels has been the use of plastic injection casts. NYSTROM (9) has employed this method in the delineation of the vasculature of gliomas as well as cerebral metastases. A distinct advantage of this technique is the outlining of arteries and veins with plastics of different colors thereby producing easy identification of the vascular components in the tumor mass. Unfortunately inspection of the photographs of the casts made by NYSTROM would seem to indicate that only large tumor vessels are filled and that a major part of the capillary circulation is not outlined. Also the digestion of surrounding cerebral tissue is time consuming and the relationship of the pathologic vessels to the tumor is lost.

The value of contrast material injection and radiographic study of pathologic intracerebral vessels has been highlighted by RUSSELL (12) in his study of small aneurysms (300 to 900 micra) arising from the lenticulostriate arteries in middle aged hypertensive and normotensive patients. Using a combination of micropaque gelatin and water RUSSELL was able to fill arterioles down to 30 micra and occasionally venous structures were visible following the passage of contrast material through the normal capillary bed.

Although the very fine particulate size of the contrast material (Thorotrast 0.1 micra and Micropaque 0.1—3.0 micra) employed in the present study is helpful in filling tumor vessels down to 10 to 25 micra there are other important factors that contribute to the success of the method. In many of the injections there appeared to be a preferential filling of tumor vessels by the contrast agent. It must be assumed and as pointed out by HARDMAN (6), that

amus. Because of the sharp delineation and almost homogeneous appearance a large metastasis was favored prior to death. A diffuse irregular meshwork of capillaries was found throughout the tumor (Fig 2a) with accompanying spotty areas of central necrosis. Dilatation of the hantulostrite arteries supplying the tumor as well as the irregular distribution of necrotic areas in the tumor was well shown in a microradiogram (Fig 2b) of an 8 mm thick section taken from the coronal section shown in Fig 2a. A 10 micra histological section (Fig 2c) from the same tumor area showed that capillaries down to 10–15 micra were filled with contrast agent without evidence of extravasation. The microscopic diagnosis of the tumor was astrocytoma grade IV.

Case 3 This 48 year old woman had become disoriented over an eight week period. On admission a moderate right hemiparesis and right facial weakness was found. Left carotid angiography revealed elevation and stretching of vessels of the middle cerebral group and a faint small contrast material stain was seen in the late arterial phase (Fig 3a). The radiologic diagnosis was a probable astrocytoma with cyst formation although a small aneurysm with an associated intracerebral hematoma could not be excluded. A cyst was tapped containing xanthochromic fluid. At post mortem only the left cerebral hemisphere was injected and a roentgenogram of the coronal section bearing a solid part of the tumor showed the tumor vessels to be well filled (Fig 3b). In several areas the vessels appeared to be arranged in rounded clusters. Microradiography of one of these formations (Fig 3c) measuring approximately 3×6 mm showed an intricate network of vessels interspersed between viable and necrotic tumor. Histologic sections showed astrocytoma grade III. The vascular elements in the formation shown consisted of arterioles, dilated capillaries and venules with thickened muscular walls indicating a well formed arteriovenous shunt.

Case 4 This 42 year old male had severe frontal headaches for three weeks prior to admission. EEG suggested a right frontotemporal lesion and a right carotid angiography showed extensive pathologic vascularity in the right frontal lobe consistent with an advanced glioblastoma. At surgery only partial removal of the right frontal lesion could be carried out. Coronal section roentgenograms showed marked tumor vascularity remaining in the right frontal lobe. Extension of tumor with its associated vascular supply to the opposite hemisphere via the corpus callosum had occurred. A microradiogram (Fig 4) showed prominent arteriovenous connections of the tumor mass in the corpus callosum and septum pellucidum. Histologic sections revealed astrocytoma grade IV.

Case 5 This 54 year old male exhibited signs of progressive confusion over a three week period. A chest film showed a mass in the left hilum. Bilateral carotid angiography was negative. At post mortem multiple cerebral metastases were found which had originated from a lung carcinoma. The largest deposit noted which was a little over 1 cm in diameter was well filled with the contrast agent as shown in coronal section radiography (Fig 5a). A microradiogram (Fig 5b) of a 3 mm thick section through this lesion revealed a conglomeration of tumor vessels with an area of central necrosis and some extravasation of contrast material.

Discussion

During the last thirty five years several techniques have been developed for the post mortem evaluation of pathologic vessels in the brain. The foremost of these has been the use of erythrocyte stains. The interaction of hemoglobin



a



b

Fig 5 Case 5 a) Roentgenogram of 1.5 cm coronal section bearing well vascularized metastasis (arrow) from lung carcinoma b) Microradiogram ($\times 7$) of metastasis in Fig 5 Section 3 mm thick closely packed capillaries around area of central necrosis Extra collection of contrast in area hemorrhage in tumor

generated by low voltage (10–20 kV) microradiographic units, as well as the use of fine grain photographic emulsions are primarily responsible for the good resolution of small vessels. This is well shown by examination of Fig 1 b and c. The coronal section radiogram in Fig 1 b was produced by standard roentgen film at 50 kV and 10 mAs. Although the tissue thickness of this specimen is approximately twice that of the microradiographic section (Fig 1 c) the resolution of vascular detail in the region of the arrow (Fig 1 b) is considerably less when the two films (without magnification) are compared. It is also of interest that photographic enlargement of specimens roentgen examined with industrial film as suggested by McALISTER (8) does not appear to give the same vascular detail as obtained with microradiography.

The time required to produce microradiograms is surprisingly short. With the unit described, an exposure of approximately 1 minute per millimeter of tissue thickness is required at 20 kV and 10 mA. Photographic enlargement of the microradiogram is carried out easily and vascular detail can be further improved when television amplification (LogEtronic) methods are used.

HARDMAN (6), in his description of the angioarchitecture of gliomas with a high grade of malignancy suggested that each had a large area of central necrosis surrounded by a narrow ring of tumor containing capillaries and sinusoids. He distinguished four vascular zones around and in the lesion. The outer zone (Zone 1) in the surrounding white matter exhibited either a normal capillary network or arterioles and capillaries compressed by adjacent



Fig. 4 Case 4. Microradiogram ($\times 2$) of tumor vascularity of transfrontal astrocytoma. Thickness of section 6 mm. Tributaries from pericallosal artery as well as dilated capillaries of tumor in corpus callosum and septum pellucidum are seen. Contrast material also fills the septal veins.

dilated vessels supplying the tumor as well as arteriovenous connections, particularly within the glioblastomas, provide areas of decreased vascular resistance and this permits more thorough filling of tumor vessels with contrast agent. It is estimated that some of the injections outlined as much as 75% of the vascular bed including tumor capillaries, although in general the success rate ranges between 40—50%.

Another factor which expedites vessel filling is the nearly intact state of the tumor vessels at the time of autopsy. The majority of brains injected had either no previous surgical transformation or were subjected to diagnostic biopsy only. In the biopsied brains, although bleeding did occur at the biopsy site, it was found at post mortem that the central, and usually necrotic portion of the tumor, was hemorrhagic and a considerable portion of the vasculature in the peripheral viable areas of the tumor was not distorted. Of interest is the fact that surprisingly little extravasation of contrast material occurred in the tumors. This can probably be accounted for again on the basis of decreased vascular resistance in the tumor and the intact state of the tumor vessels at autopsy.

Microradiography aids considerably in the assessment of the angioarchitecture of normal and pathologic tissue. The clarity of injected vessels, particularly at a capillary level, has been well shown by several authors including BARCLAY (1), CHANG (2) and PEREZ (10). The longer x-ray wave lengths



Fig 5 Case 5 a) Roentgenogram of 1.5 cm coronal section bearing well vascularized metastasis (arrow) from lung carcinoma b) Microradiogram ($\times 7$) of metastasis in fig 5a Section 3 mm thick closely packed capillaries around area of central necrosis Extra vasation of contrast in area hemorrhage in tumor

generated by low voltage (10—20 kV) microradiographic units as well as the use of fine grain photographic emulsions are primarily responsible for the good resolution of small vessels. This is well shown by examination of Fig 1 b and c. The coronal section radiogram in Fig 1 b was produced by standard roentgen film at 50 kV and 10 mAs. Although the tissue thickness of this specimen is approximately twice that of the microradiographic section (Fig 1 c) the resolution of vascular detail in the region of the arrow (Fig 1 b) is considerably less when the two films (without magnification) are compared. It is also of interest that photographic enlargement of specimens roentgen examined with industrial film as suggested by McALISTER (8) does not appear to give the same vascular detail as obtained with microradiography.

The time required to produce microradiograms is surprisingly short. With the unit described an exposure of approximately 1 minute per millimeter of tissue thickness is required at 20 kV and 10 mA. Photographic enlargement of the microradiogram is carried out easily and vascular detail can be further improved when television amplification (LogEtronic) methods are used.

HARDMAN (6) in his description of the angioarchitecture of gliomas with a high grade of malignancy suggested that each had a large area of central necrosis surrounded by a narrow ring of tumor containing capillaries and sinusoids. He distinguished four vascular zones around and in the lesion. The outer zone (Zone A) in the surrounding white matter exhibited either a normal capillary network or arterioles and capillaries compressed by adjacent

tumor. In some sections dilated capillaries and venules were distinguished. The outer zone of vessels (Zone B) in the viable tumor border consisted of capillaries which were irregularly arranged and dilated. The inner zone of vessels (Zone C) in the active portion of the tumor were interpreted as sinusoids and these were found to be generally larger than the capillaries in Zone B. The necrotic center of the tumor was designated as Zone D and the vascular elements found were primarily thrombosed sinusoids.

Although evaluation of the gliomas injected in the present survey is not yet complete, it would seem that HARDMAN's analysis can provide only a stereotype of the vascular anatomy of these tumors. Examination of the micro radiograms of the tumors in the first three patients (Figs 1 c, 2 b and 3 c) indicates that considerable variation in vascular formation is present.

In Case 1 (Fig. 1 c) the vascular border of the tumor is well defined between the central area of necrosis and normal brain. A variation in caliber of vessels is present throughout the viable tumor border and division of vessels into zones, i. e., peripheral small capillaries and inner dilated sinusoids is not possible. In this case, although widened tortuous channels (sinusoids) could be seen in several sites along the necrotic margin of the tumor, for the most part, thrombosed capillaries which have not undergone dilatation end abruptly in the necrotic portion of the lesion. The abrupt ending of capillaries without deformation by the necrotic portion of the tumor is well shown in Case 2 (Fig. 2 b), and this configuration has been noted in the majority of the gliomas examined at present. Possibly, a rapid rate of tumor growth in the more malignant lesions does not permit the development of drainage channels on the inner aspect of the viable tumor border. In all probability, the capillary bed in this area is engulfed rapidly by the necrotic debris of the tumor. In Case 3 (Fig. 3 c), on the other hand, well preserved networks of vessels were noted throughout the tumor mass. Histologic sections showed well developed arterio-venous shunts with evidence of muscular hypertrophy along the walls of draining venules indicating the presence of increased blood flow.

It is interesting to note that Cases 1 and 3 were both graded as astrocytoma grade III and yet a markedly different angio architecture is present in the lesions. In Case 1 the vascular structures appear to support the growth of the viable edge of the tumor without an organized pattern. There is no specific vascular arrangement other than a circumferential one and this appears to be dictated by the expanding necrotic center of the tumor.

In Case 3, although a large center of liquefactive necrosis was present, well developed and repetitive constellations of vessels were seen. The reasons for this disparity are not yet apparent in our studies. Possibly, although the lesions have the same grade of malignancy, Case 3 was a more slowly growing tumor.

and more time was available for the tumor mass to prepare a well organized vascular bed. Perhaps a greater understanding of this problem can be gained by the study of the angio architecture of gliomas transplanted to laboratory animals. GREENE (5) has shown that human brain tumors can be transplanted to guinea pig and mouse brains with subsequent varying rates of tumor growth. Injection studies in these animals and histological correlation may give some insight concerning the development of tumor angioarchitecture in relation to tumor growth as well as vascular formations in varying types and grades of malignancy.

Some preliminary observations may be made regarding the correlation of cerebral angiographic tumor stains and the same tumor angioarchitecture outlined by injection at post mortem. It would appear that when prominent central necrosis is present in a glioma a critical thickness of tumor capillaries and sinusoids must be present before a recognizable angiographic stain is seen. In several cases studied although an obvious displacement of major or secondary vessels was present no tumor stain could be seen. The post mortem injection roentgenograms and histologic confirmation revealed a fringe of contrast material filled vessels usually less than 1/2 cm wide.

It is also of interest to note that in two brains examined with multiple metastases from carcinoma of the lung bilateral cerebral angiographies were carried out and no contrast filling of tumor vessels could be seen. The metastases were generally 1 cm or less in diameter and the majority of these were well filled with contrast material (see Fig 5 b) during post mortem injection. Although it is well known that metastatic lesions may not be seen during cerebral angiography the findings in this study underline the fact that the size of the lesion is important in determining the presence of an *in vivo* angiographic stain.

Acknowledgements

The authors wish to thank Mr Wylie McKussock for his permission to initiate and carry this project forward at the Atkinson Morley's Unit St George's Hospital London. Thanks are also extended to Doctor G. B. Solitare of the Department of Neuropathology at the Yale Medical Center and Dr G. I. DeSuto-Nagy of the Department of Pathology at the West Haven Connecticut Veterans Hospital for their help in the analysis of the pathologic material. The study was supported by Grant (BT 1050) U. S. Public Health Service.

SUMMARY

A post mortem brain tumor injection technique employing a mixture of Thorotrast and Micropaque is described by which 50 to 75 % of the tumor circulation at a capillary level can be outlined. Considerable variation was noted in the size, number and configuration

of vascular elements throughout the viable sections of gliomas. In order to obtain *in vivo* angiographic identification of a glioma with prominent central necrosis it would appear that the tumor must have a vascular border 1/2 cm or greater in thickness. Well vascularized metastases under 1 cm in diameter could not be demonstrated during cerebral angiography.

ZUSAMMENFASSUNG

Es wird die Technik einer Injektion eines Hirntumors post mortem mit einer Mischung von Thorotrast und Micropaque, womit 50—75 % der Tumorgefässe dargestellt werden können, beschrieben. Die Gefäss Elemente in den viablen Teilen von Gliomen variierten bedeutend in ihrer Grösse, Anzahl und Form. Um eine angiographische Darstellung eines Glioms mit bedeutender zentraler Nekrose *in vivo* zu erhalten, muss der Tumor allem Anschein nach ein Mindestmass von 1/2 cm Dicke haben. Gut vaskularisierte Metastasen unter 1 cm Durchmesser konnten mittels cerebraler Angiographie nicht zur Darstellung gebracht werden.

RÉSUMÉ

Description d'une technique d'injection post mortem des tumeurs cérébrales par un mélange de Thorotrast et de Micropaque qui permet de remplir 50 à 75 % de la circulation tumorale à l'étage capillaire. Les auteurs ont constaté des variations considérables dans le calibre, le nombre et la configuration des éléments vasculaires dans les parties viables des gliomes. Pour qu'on puisse identifier *in vivo* par angiographie un gliome présentant une importante nécrose centrale, il semble qu'il faut que la tumeur ait une coque vascularisée ayant au moins un demi centimètre d'épaisseur. Il n'a pas été possible de mettre en évidence par angiographie cérébrale des métastases bien vascularisées de moins d'un centimètre de diamètre.

REFERENCES

- 1 BARCLAY A F and BENTLEY F H. The vascularization of the human stomach. A preliminary note on the shunting of trauma. *Brit J Radiol* 32 (1949) 62.
- 2 CHANG C H and TREMBLEY B. Microangiographic studies of kidney and brain in animals. Technical aspects. *Yale J Biol Med* 33 (1961) 451.
- 3 COURVILLE C H. Vascular pattern of the encephalic gray matter in man. *Bull Los Angeles neurol Soc* 23 (1958) 30.
- 4 EROS G. Method for fuchsin staining of the network of cerebral blood vessels. *Arch Path* 31 (1941) 215.
- 5 GREENE H S N. The transplantation of human brain tumors to the brains of laboratory animals. *Cancer Res* 13 (1953) 422.
- 6 HARDMAN J. The angio architecture of the gliomata. *Brain* 63 (1940) 91.
- 7 KERNOHAN J W, MAHON R F, SVEN H J and ANDSON A W. A simplified classification of the gliomas. *Proc Mayo Clin* 24 (1949) 71.
- 8 McALISTER W H, MARGULIS A R, HEINBECKER P and SEJUT H. Arteriography and microangiography of gastric and colonic lesions. *Radiology* 79 (1962) 769.

- 9 NYSTROM S Pathological changes in blood vessels of human glioblastoma multiforme
Acta path microbiol scand (1960) Suppl No 137
- 10 PEREZ C A HODGES F J and MARGULIS A R Microradiography in experimental
cerebral edema in rats Radiology 82 (1964) 529
- 11 PICKWORTH F A A new method of study of the brain capillaries and its application
to the regional localization of mental disorder J Anat 69 (1934—35) 62
- 12 RUSSELL R W R Observations on intracerebral aneurysms Brain 86 (1963) 425

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REFERENCES

1. BARGLEY A. F. and BENFELD L. H. The vascularization of the human stomach. A preliminary note on the shunting of trauma. *Brit. J. Radiol.* 32 (1949) 62.
2. CHANG C. H. and TRIMBLEY B. Microangiographic studies of kidney and brain in animals. Technical aspects. *Yale J. Biol. Med.* 33 (1961) 451.
3. COURVILLE C. B. Vascular pattern of the encephalic gray matter in man. *Bull. Los Angeles neurol. Soc.* 23 (1958) 30.
4. FROS G. Method for fuchsin staining of the network of cerebral blood vessels. *Arch. Path.* 31 (1941) 215.
5. GREENE H. S. N. The transplantation of human brain tumors to the brains of laboratory animals. *Cancer Res.* 13 (1953) 422.
6. HARDMAN J. The angio architecture of the gliomata. *Brain* 63 (1940) 91.
7. KERNOHAN J. W., MAHON R. F., SALEN H. J. and ADSON A. W. A simplified classification of the gliomas. *Proc. Mayo Clin.* 24 (1949) 71.
8. MCALISTER W. H., MARCUS A. R., HEINBECKER P. and SEJUT H. Arteriography and microangiography of gastric and colonic lesions. *Radiology* 79 (1962) 769.

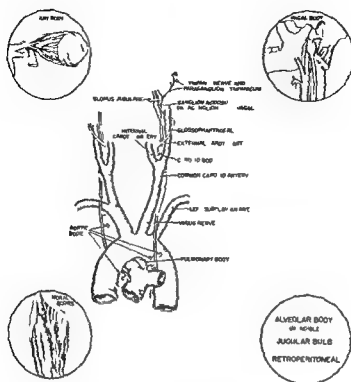


Fig 1 Sites of chemoreceptor tissue (after LeCompte)

ogy and behavior of this tumor and have led to a multitude of terminologies. The term carotid body tumor familiar to all is slowly losing favor to the more appropriate chemodectoma (MULLIGAN 1950) which is receiving increased recognition (Editorial J Amer med Ass 1964). The carotid body was discovered by Von HALLER in 1743 and in 1891 MARCHAND first described a case of a carotid body tumor. RIEGNER removed the first carotid chemodectoma in 1880 (BYRNE 1953).

There are a number of histologically similar structures located in various parts of the body and aggregates of such tissue have been described at other sites in addition to the carotid bifurcation and the jugular foramen. Since the chemoreceptor tissue at these various sites has been assumed to be similar a classification may be based on anatomic location (BOYD 1937 see Fig 1).

Rare tumors arise from the vagal body (BURMAN 1955 and 1956), aortic body (LATTES 1950, DUNCAN & McDONALD 1951, McDONALD 1956), ciliary body (FISHER & HAZARD 1952, LATTES, McDONALD & SPROUL 1954), mandible (BURMAN 1956), femoral body (SMETANA & SCOTT 1951).

FROM THE NEURORADIOLOGICAL UNIT (CHIEF M M SCHECHTER) OF THE DEPARTMENT OF RADIOLOGY (DIRECTOR F RUZICKA) AND THE NEUROLOGY SERVICE (CHIEF J ■ CHUSID), ST VINCENT'S HOSPITAL AND MEDICAL CENTER OF NEW YORK, AND FROM THE DIVISION OF NEURORADIOLOGY (CHIEF M M SCHECHTER) OF THE DEPARTMENT OF RADIOLOGY (DIRECTOR M ELKIN), ALBERT EINSTEIN COLLEGE OF MEDICINE, NEW YORK, U S A

CHEMODECTOMAS OF THE CAROTID BIFURCATION

by

MANNIE M SCHECHTER and JOSEPH G CHUSID

The two carotid bodies are highly vascularized chemoreceptor organs located at the carotid bifurcations and important in the reflex control of respiration. Each carotid body (glomus caroticum) contains islands of chemoreceptor cells and sustentacular cells surrounded by sinusoidal vessels (Ross 1959). Afferent nerve fibres from the carotid bodies ascend to the medulla via a branch of the glossopharyngeal nerve (nerve of Hering). Experimental studies on animals, initially undertaken by HEDGECOCK and coworkers (1930) have shown that the carotid bodies are sensitive to oxygen deficiency (decline in partial pressure of arterial oxygen), excess carbon dioxide (rise in partial pressure of arterial carbon dioxide) or increased acidity (lower arterial pH). Under these conditions of carotid body stimulation, the animal responds by hyperventilation with increased rate and amplitude of respiration (OBERHOLZER & TOFANI 1960, ULLAS 1960, ANICHKOV & BELENKH 1963, GANONG 1963). The functional significance of the carotid bodies in man has been assumed to be similar to that so clearly demonstrated in lower animals, but confirmation of the precise role of carotid bodies in human physiology must await further studies.

Conflicting opinions appear in the literature concerning the function, histol

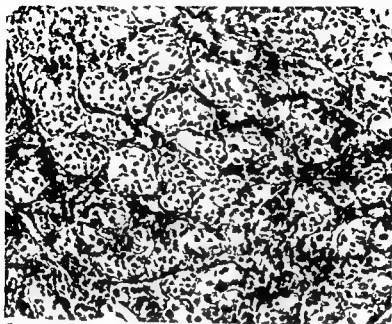


Fig 2 Case 5 Histologic appearance showing nests of cells (Zellballen) Hematoxylin and eosin 250

Chemodectomas are usually encapsulated globular and red brown or red gray in appearance. Their consistency may vary from firm to soft and on cross section they appear homogeneous. Tumor size usually ranges from 2 to 5 cm in diameter. They nestle in the carotid bifurcation. A plane of cleavage may be present between these tumors and the vessels or they may be intimately bound to the carotid arteries (BIRGE 1958).

Histologically nests or alveolar clusters of epithelioid cells (Zellballen) are separated by a vascular fibrous stroma. LECOMPTE (1951) felt that these tumors may be divided into three groups: 1) the usual or commonest type in which the normal carotid body structure is reproduced faithfully; 2) the adenoma like type in which the chief cells are rounded plump with abundant cytoplasm resembling epithelium supported by scanty stroma; and 3) the angioma like type in which chief cells have spindle or crescent shapes simulating endothelial cells. Reticulum stains may help in demonstrating relationship between cell clusters and surrounding stroma.

Variation in microscopic appearance may be noted with some areas appearing more angiomatous than others. However a correlation between the histologic appearance of chemodectomas and their behaviour has not been firmly established. There is no evidence that carotid chemodectomas are

RANDALL & WALTER 1954), and retroperitoneal body (SMETANA & SCOTT 1951, AREAN & DEARELLANO 1956)

Although over 500 carotid chemodectomas have been reported, many questions relating to these tumors remain unanswered and conflicting opinions appear in the literature. Since the majority of these tumors are benign and slow growing with minimal associated symptoms, surgical intervention is not considered justifiable unless the tumor can be removed without sacrificing the internal carotid artery in the neck (McILRATH & REMINE 1963), or compromising its flow (CONLEY 1963). These tumors are situated in a critical anatomic site, the carotid bifurcation, and the chemodectoma often encompasses the common, internal and external carotid arteries. Should the carotid artery be inadvertently interrupted in the neck, prior knowledge of the collateral supply to the brain from the opposite side would be useful.

Angiography may help in establishing the diagnosis when a mass in the neck is present. It is surprising to find that relatively few references have appeared in the literature on the use of angiography in the evaluation of neck tumors. It is our purpose to emphasize the value of carotid angiography in the diagnosis of carotid bifurcation chemodectomas, and to report 13 cases of carotid chemodectomas.

Most patients with chemodectomas of the carotid bifurcation are asymptomatic except for the presence of a painless cervical mass. Growth of the tumor is usually quite slow and its discovery may be delayed until it has attained massive size. In some instances, the cervical mass may be detected only as an incidental finding on routine physical examination. These tumors may be present for several years and in one of our cases was known to the patient for twenty five years. When the tumor becomes quite large, the patient may become aware of the mass and perhaps of some local discomfort. Occasionally in masses which grow medially and project into the pharynx, dysphagia may be associated.

On physical examination, the chemodectoma is usually palpable as a firm mass in the cervical region near the carotid bifurcation and usually is movable laterally but not vertically. Often a slight, superficial neck swelling or bulge may be noted over the tumor area. The tumor may be pulsatile and have associated bruit or thrill, making clinical distinction from aneurysm difficult. Intrioral examination may disclose bulging of the oropharyngeal wall in those patients whose tumors project into this region. MARTORELL (1956) claimed that pressure over the common carotid artery proximal to a carotid chemodectoma could result in diminution in the size of the tumor which would gradually assume its normal size after compression was released, aneurysms, he stated, usually assumed precompression size rapidly after release of pressure.



Fig 2 Case 5 Histologic appearance showing nests of cells (Zellballen) Hematoxylin and eosin, $\times 250$

Chemodectomas are usually encapsulated globular and red brown or red gray in appearance. Their consistency may vary from firm to soft and on cross-section they appear homogeneous. Tumor size usually ranges from 1 to 3 cm in diameter. They nestle in the carotid bifurcation. A plane of cleavage may be present between these tumors and the vessels or they may be intimately bound to the carotid arteries (BYRNE 1958).

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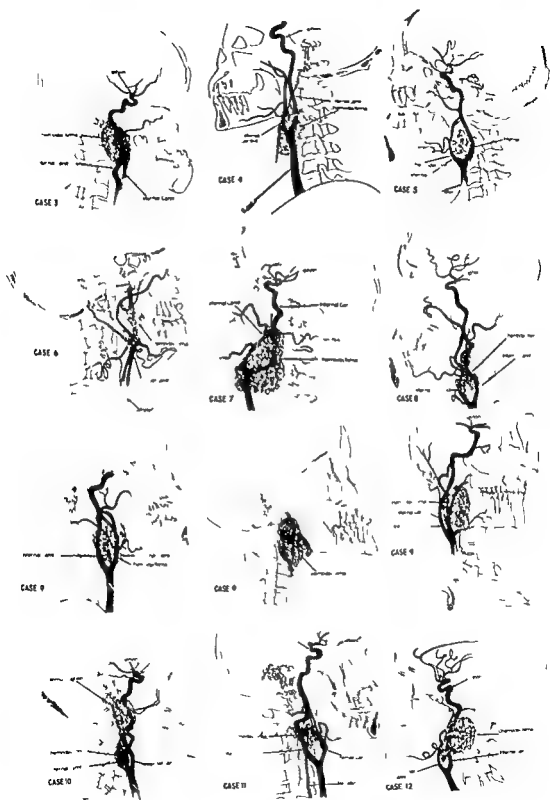


Fig. 3 For text see opposite page

more responsive or sensitive to blood changes than normal carotid bodies. Although the vast majority are clinically benign, metastases to regional lymph nodes or other distant sites have been reported. This did not occur in our series.

In most large series such as the Mayo Clinic's 64 cases (McILRATH & REVINE 1963) it has been noted that the majority of chemodectomas of the carotid bifurcation occur unilaterally. Although these tumors are usually unilateral, bilateral tumors are by no means rare (HARRINGTON, CLAGETT & DOCKERTY 1941; LAHEY & WARREN 1951; BIRRELL 1952; PETTET, WOOLNER & JUDD 1953; ENGSTROM & HANBERGER 1957; BYRNE 1958; SESSIONS, MCSWAIN, CARLSON & SCOTT 1959; WESTBURY 1960; RUSH 1962; CONLEY 1963; MORRIS et coll 1963; REESE, LUCAS & BERGMAN 1963; KROLL et coll 1964). In the series by REESE et coll (1963) four of twelve cases had bilateral tumors. Two cases in our series (nos. 10 and 12) had bilateral carotid chemodectomas; in one the second tumor appeared after a nine year period and in the other after a ten year period. One patient (Case 10) had in addition a chemodectoma of the glomus jugulare.

These tumors are more common (17/1) in women (McILRATH & REVINE). A wide age range, 6 months (WOSNESSENAI — cited by TAYLOR 1940) to 80 years (BYRNE 1958) and a long history of tumor existence, 2 months to 36 years have been reported. Concurrent associations, i.e. multicentricity of carotid and glomus jugulare chemodectomas have been reported (HARRINGTON, CLAGETT & DOCKERTY 1941; KIPKIE 1947; ASKENASY, EPPENSTEIN & HERZBERGER 1953; SCHADE 1953; HAWKINS 1961; CONLEY 1963). This was present in Case 10 in our series.

A heredo-familial incidence has been claimed by LEWISON & WEINBERG (1950); LAHEY & WARREN (1951); PETTET, WOOLNER & JUDD (1953); DESAI & PATEL (1961); LADENHEIM & SACHS (1961); RUSH (1962), and CONLEY (1963). CHASE (1933) reported a tumor in two sisters and LAHEY & WARREN (1947) reported tumors in three siblings of one family and two other members of the same family had excision of the tumors by other surgeons. KROLL et coll (1964) report twelve individuals in two generations with carotid chemodectomas; of these twelve, five had tumors on both sides.

The vast majority of these tumors are benign and even in malignant tumors mitotic figures are rare. MIROV (1962) reported a case of a malignant carotid chemodectoma with regional invasion and metastasis. A case with wide spread pulmonary metastases proven by lung biopsy was reported by FANNING, WOODS & CHRISTIAN (1963) and the patient remained symptom free for over

Fig. 3. Scheme in our presentation of the chemodectomas of the carotid bifurcation demonstrated by angiography in our cases (Case 13 not included).

two years TURNBULL (1954) also reported a case with metastatic spread to lungs. Bone metastases have been reported by ANDERSON & SCARCELLA (1963). DONALD & CRILE (1948) reported a case with spread to pelvis, ribs, femur and sternum. GOODOF & LISCHER (1943) reported a case with spread to pancreas, and SPOTNITZ (1951) had a case with spread to cervical glands, skin, kidney, pancreas, heart and pleura. Other visceral metastases reported include GRONBERGER (1917) spread to kidneys and mediastinum and GILFORD & DAVIS (1904) spread to liver. Other reports of malignancy include MORFIT, SWAN & TAYLOR (1953), ROMANSKI (1954), WARREN (1959), MODERG (1961), REESE, LUCAS & BERGMAN (1963) and MORRIS et coll (1963).

LE COMPTE (1951) stated that he had never seen a malignant carotid chemolectoma. In one case in his series, a node enclosed near the tumor was involved, but this could have been due to direct extension. In a case reported by RUSH (1962), local invasion was encountered in the brachial plexus. REESE et coll (1963) report 13 instances of regional lymph node involvement and 15 instances of distant dissemination. MACCOMB (1948) reported recurrence in nodes in the neck after resection of tumor and AREAN (1955) reported tumor extending into the spinal canal. Spread to adjacent lymph glands has also been reported by SHOWAN & OWEN (1938), LAHEY & WARREN (1947), MONRO (1950), GOARMAGHTICH & PATTAN (1954), FRIEDMAN & LAU (1957), RABSON & ELLIOT (1957), and FERNAN ZEGARRA (1957).

Radiology

Apart from a soft tissue mass in the conventional film of the neck, no special changes have been reported in the literature or in our group. The changes due to metastases have been excluded. Negative or normal findings such as normal intervertebral foramina, and absence of bone erosion are usually observed.

Carotid angiography: Despite some of the unique physical findings, a correct preoperative diagnosis is often not made according to most authors. LAHEY & WARREN (1951) reported 18 patients in whom a correct diagnosis was made in only 9 cases (quoting WETZEL). In the series of 5 cases reported by HAWKINS (1961) a correct diagnosis was made only after carotid angiography. In WESTBURY's series (1960) in none of seven cases was a preoperative diagnosis made by the referring surgeon. Bilateral angiography performed in three of the cases showed characteristic changes. However, MONRO (1950) stated that angiography would appear to play no part in carotid body tumor diagnosis unless it be in the occasional case where the possibility of aneurysm can not be excluded on clinical grounds.

Angiography is a most valuable diagnostic aid in the study of carotid chemodectomas (CONLEY 1963). It is usually diagnostic for the medium and the large carotid chemodectomas and is highly suggestive for the smaller ones. MORRIS et coll. (1963) claimed that angiography provided the most accurate diagnosis before surgery. Angiography may establish a diagnosis and may also supply other information such as the size of the tumor, its spatial relationship to the common internal and external carotid arteries and a collateral flow from the opposite side via the circle of Willis or extracranial anastomotic channels. Lesions higher in the neck and unsuspected lesions on the opposite side may be revealed (ENGSTROM & HANBERGER 1957).

The first angiographic study of these tumors was reported by LICHTENAUER (1938) following tumor biopsy. Since then many reports have appeared (IDDOHRY 1951, WRIGHT 1952, LIPSCHITZ 1958, BORELLI 1959, MYBURGH & BERK 1959, NELSON 1962 and others).

Carotid angiography may be performed by puncturing the common carotid artery directly or introducing a catheter into this vessel from another site of puncture. Selective injection into the internal and external carotid arteries may be used. When a large mass is encountered the carotid artery may be punctured in a retrograde direction. A dosage of 8–10 ml of contrast medium is usually sufficient. Serial films should be taken to demonstrate the filling and emptying of the tumor with contrast medium. Carotid angiography of the contralateral side with compression on the carotid artery of the affected side should be performed to demonstrate patency of collateral channels should it become necessary to sacrifice the involved carotid artery.

The most common finding on angiography is contrast filling of the vascular tumor mass. A well circumscribed accumulation of contrast medium within a tangle of regular vessels almost giving the impression of homogeneous filling of the mass is seen (Figs 4, 5). It does not however approach the dense, completely homogeneous filling as in an aneurysm of the carotid artery. Since the tumor often nestles between the internal and external carotid arteries these arteries are displaced in a characteristic way. The tumor mass is always situated at the bifurcation and receives most of its blood supply from the external carotid artery. The vessels embraced by the tumor may be slightly narrowed but are never significantly occluded. The common carotid artery proximal to the tumor may be hypertrophied (Figs 3, 5, 6).

Carotid angiographies were performed on twelve of the thirteen cases in our series (see Table). In eleven cases the characteristic changes were present, in the twelfth case the carotid vessels had been previously ligated and an injection on the normal side failed to fill the tumor side. The angiographic preoperative diagnosis was correct in all cases.

two years TURNBULL (1954) also reported a case with metastatic spread to lungs. Bone metastases have been reported by ANDERSON & SCARCELLA (1963). DONALD & CRILE (1948) reported a case with spread to pelvis, ribs, femur and sternum. GOODOR & LISCHER (1943) reported a case with spread to pancreas, and SPOTNITZ (1951) had a case with spread to cervical glands, skin, kidney, pancreas, heart and pleura. Other visceral metastases reported include GRONBERGER (1917) spread to kidneys and mediastinum and GILFORD & DAVIS (1904) spread to liver. Other reports of malignancy include MORFIT, SWAN & TAYLOR (1953), ROMANSKI (1954), WARREN (1959), MODERG (1961), REFSF, LUCAS & BERGMAN (1963) and MORRIS et coll (1963).

LE COMITE (1951) stated that he had never seen a malignant carotid chemodectoma. In one case in his series, a node enclosed near the tumor was involved, but this could have been due to direct extension. In a case reported by RUSH (1962), local invasion was encountered in the brachial plexus. REESE et coll (1963) report 13 instances of regional lymph node involvement and 15 instances of distant dissemination. MACCOMB (1948) reported recurrence in nodes in the neck after resection of tumor and AREAN (1955) reported tumor extending into the spinal canal. Spread to adjacent lymph glands has also been reported by SHOWAN & OWEN (1938), LAHEY & WARREN (1947), MONRO (1950), GOARMAGITICH & PATTIN (1954), FRIEDMAN & LAU (1957), RANSON & ELLIOT (1957), and FERNAN ZEGARRA (1957).

Radiology

Apart from a soft tissue mass in the conventional film of the neck, no special changes have been reported in the literature or in our group. The changes due to metastases have been excluded. Negative or normal findings such as normal intervertebral foramina, and absence of bone erosion are usually observed.

Carotid angiography. Despite some of the unique physical findings, a correct preoperative diagnosis is often not made according to most authors. LAHEY & WARREN (1951) reported 18 patients in whom a correct diagnosis was made in only 9 cases (quoting WETZEL). In the series of 5 cases reported by HAWKINS (1961) a correct diagnosis was made only after carotid angiography. In WESTBURY's series (1960), in none of seven cases was a preoperative diagnosis made by the referring surgeon. Bilateral angiography performed in three of the cases showed characteristic changes. However, MONRO (1950) stated arteriography would appear to play no part in carotid body tumor diagnosis unless it be in the occasional case where the possibility of aneurysm can not be excluded on clinical grounds.

Angiography is a most valuable diagnostic aid in the study of carotid chemodectomas (CONLEY 1963). It is usually diagnostic for the medium and the large carotid chemodectomas and is highly suggestive for the smaller ones. MORRIS et coll (1963) claimed that angiography provided the most accurate diagnosis before surgery. Angiography may establish a diagnosis and may also supply other information such as the size of the tumor, its spatial relationship to the common internal and external carotid arteries and a collateral flow from the opposite side via the circle of Willis or extracranial anastomotic channels. Lesions higher in the neck and unsuspected lesions on the opposite side may be revealed (ENGSTROM & HANBERGER 1957).

The first angiographic study of these tumors was reported by LICHTENAUER (1938) following tumor biopsy. Since then many reports have appeared (ISDOHRN 1951, WRIGHT 1952, LIPSCHITZ 1958, BORELLI 1959, MYBURGH & BERA 1959, NELSON 1962 and others).

Carotid angiography may be performed by puncturing the common carotid artery directly or introducing a catheter into this vessel from another site of puncture. Selective injection into the internal and external carotid arteries may be used. When a large mass is encountered, the carotid artery may be punctured in a retrograde direction. A dosage of 8–10 ml of contrast medium is usually sufficient. Serial films should be taken to demonstrate the filling and emptying of the tumor with contrast medium. Carotid angiography of the contralateral side with compression on the carotid artery of the affected side should be performed to demonstrate patency of collateral channels should it become necessary to sacrifice the involved carotid artery.

The most common finding on angiography is contrast filling of the vascular tumor mass. A well circumscribed accumulation of contrast medium within a tangle of regular vessels almost giving the impression of homogeneous filling of the mass is seen (Figs 4–5). It does not however approach the dense completely homogeneous filling as in an aneurysm of the carotid artery. Since the tumor often nestles between the internal and external carotid arteries these arteries are displaced in a characteristic way. The tumor mass is always situated at the bifurcation and receives most of its blood supply from the external carotid artery. The vessels embraced by the tumor may be slightly narrowed but are never significantly occluded. The common carotid artery proximal to the tumor may be hypertrophied (Figs 3–5–6).

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Table

Data concerning twelve patients examined by carotid angiography

Case no.	Sex	Age	Side of tumor	Length of history (yrs)	Symptoms
1	F	58	Left	12	Mass left neck gradually increasing in size
2	F	23	Left	7	Enlarging mass in left neck
3	F	48	Right	1/2	Swelling of right upper neck
4	F	32	Left	1/4	Mass left neck increasing in size
5	F	33	Left	3	Small gland
6	M	29	Right	1	Painless mass in neck
7	M	61	Left	25	Painless mass increasing in size
8	M	41	Right	1	Painless mass in right neck
9	F	23	Left	1	Mass left neck
10	F	34	Right	1	Painless mass in right neck
11	M	45	Right	1/2	Small mass in right neck
12	F	51	Left	18	Painless mass in left submandibular
13	M	57	Right	3/4	Painless right submandibular mass Proxymal hypertension 14 years

Case reports

Case 1 This 58 year old woman had been aware of a slowly enlarging painless mass in the left neck for about twelve years. Operation elsewhere one year previously had revealed a mass adherent to the carotid vessels which could not be completely excised and which biopsy disclosed to be a chemodectoma. Physical examination was essentially negative except for the presence of an operative scar in the left cervical region overlying a small firm mass. Diagnostic studies including roentgen examination of the chest and skull and an

Table (cont.)

Clinical diagnosis	Angiographic diagnosis	Treatment
Carotid chemodectoma	No angiogram	Excision
Bopsy previously		
Carotid chemodectoma (Previous operation)	Rt — Norm I Lt — No filling	(1) Resected with carotid ligation 4 yrs previously (2) Excised in toto Fibrotic left internal and external carotid
Aspiration biopsy	Carotid chemodectoma	No operation
Schwannoma ganglioma or carotid chemodectoma		
Previous operation	Carotid chemodectoma	No operation
Carotid chemodectoma		
Bopsy previously	Carotid chemodectoma	Excised
Carotid chemodectoma		
Bopsy previously	Carotid chemodectoma	Excised
Carotid chemodectoma		
Aneurysm	Carotid chemodectoma	No operation
Carotid chemodectoma	Carotid chemodectoma	No operation
Bopsy previously	Carotid chemodectoma	6000 R (Cobalt) previously Excised
Carotid chemodectoma	Carotid chemodectoma	(1) 9 yrs previously left carotid chemodectoma (2) 6 yrs previously mass resected (3) Right carotid chemodectoma excised
Carotid hemodectoma	Carotid chemodectoma	Excised
Carotid hemodectoma	Carotid chemodectoma	(1) 18 yrs previously right carotid chemodectoma excised (2) Left — No operation
Carotid chemodectoma	Carotid chemodectoma	Operation deferred

Case 10 had a carotid hemodectoma excised on the left side 9 years previously Six years previously a right glomus jugulare mass was resected A right carotid chemodectoma was removed on her last admission

Electrocardiograms were normal At operation a 3 x 2.5 cm mass of rubbery consistency was removed from the carotid bifurcation without complication Histologic examination revealed a chemodectoma

Case 9 A left cervical mass reported to be a chemodectoma had been removed from this 73-year-old woman at another hospital seven years prior to admission Three years thereafter



Fig. 1. Case 7. Carotid angiogram showing a chemodectoma of the carotid bifurcation in a patient previously thought to have a luetic carotid aneurysm. Extreme vascularity of the tumor.

the mass again became apparent and gradually enlarged. She complained of marked local discomfort and pain followed by headache when the tumor site was touched or compressed. A pulsatile hard mass horizontally mobile was noted close to the carotid bulb area. In the superior end of the overlying long vertical scar of the previous operation a small tender nodule was evident. Left carotid angiography revealed no filling presumably because of ligation or resection of the carotid vessels at the previous operation. A right carotid angiography was normal with little contrast entering the left anterior and middle cerebral arteries. At operation a 2.5 cm firm mass was removed from the level of the left carotid bifurcation. Tiny fibrotic left internal and external carotid arteries were noted. Histologic examination was reported as chemodectoma.

Case 3. For approximately six months this 48 year old woman had noted a painless mass in the upper right cervical region. Examination disclosed a fullness of the right upper neck with apparent displacement of the parotid gland laterally by a tumor approximately 3×4 cm involving the deep pharyngeal space at the level of the tonsil. Aspiration biopsy through the oral cavity yielded bloody fluid. The possibility of a Schwannoma, ganglioma or carotid chemodectoma was suggested because of the clinical features of the mass. Roentgen examination of the chest and skull were normal. A right carotid angiography revealed a dense homogeneous contrast filled area at the right carotid bifurcation with displacement of trachea to left by a mass. Angiographic diagnosis of a right carotid chemodectoma was made. The patient refused an operation (Fig. 3).

Case 4. This 32 year old woman had been aware of a small mass in the left upper neck for about three months. Exploratory operation had been performed by her local physician who found a markedly pulsatile mass in the left carotid bifurcation. Examination followed transfer



Fig 5 Case 8 Chemodectoma of the carotid bifurcation. Characteristic separation of the internal and external carotid arteries by the vascular tumor.



Fig 6 Case 9 Chemodectoma of the carotid bifurcation. The tumor separates the internal and external carotid arteries.

and revealed a markedly pulsatile small mass in the region of the carotid bifurcation which could be displaced horizontally but not vertically. Mild diffuse enlargement of the thyroid gland was noted. Routine studies including EEG were normal. A left carotid angiography revealed a vascular mass in the bifurcation of the carotid artery. Operation was deferred (Fig 3).

Case 5 About three years previously this 33-year-old woman consulted her physician because of an earache. Examination at that time disclosed a small left cervical gland. Subsequently operation and biopsy of this site was carried out about three months prior to admission at which time diagnosis of a carotid chemodectoma was made. Examination on admission disclosed a firm, fixed, mildly tender left submandibular mass approximately 5 cm in diameter. The skin overlying this area contained a well-healed linear scar. Roentgen examination of the skull and cervical spine were negative. EEG was normal. Left carotid angiography revealed a vascular mass at the left carotid bifurcation. At operation a very vascular soft encapsulated tumor mass (5 × 4 cm) located at the carotid bifurcation was excised. Histologic examination was reported as chemodectoma (Figs 2, 3).

Case 6 A mass in the right upper cervical region was noted five years earlier during a routine pre-employment examination of this 29-year-old man. A biopsy a year ago had disclosed the presence of a chemodectoma. Examination revealed a mild nasal quality to his voice, weakness of right lateral gaze, a bulging mass in the right lateral pharynx and mild narrowing of the right palpebral fissure. Roentgen examination of the cervical spine was negative. Chest film was normal except for tracheal deviation to the left at the base of the neck. Right carotid angiography disclosed a vascular mass in the carotid bifurcation. At operation a vascular



Fig. 7 Case 10. Chemodectoma of the right carotid bifurcation and of the right glomus jugulare. Patient had a chemodectoma of the left carotid bifurcation removed 9 years previously.

encapsulated mass approximately 6×4 cm. which extended superiorly and medially producing bulging of right lateral pharyngeal wall was excised. Histologic examination reported chemodectoma (Fig. 3).

Case 7. This 61 year old man first noted a swelling of the left neck about twenty five years previously. This was believed to be an aneurysm and attributed to syphilis which he had contracted some 18 years earlier. Repeated courses of anti-luetic therapy were given but his blood serology remained persistently positive. Two courses of roentgen treatment of the cervical mass as well as further anti-luetic drug treatment were given two years after the mass was discovered. CSF examination at that time was entirely normal. Thereafter he received further anti-luetic treatment including penicillin on several occasions, his most recent being two years prior to admission. The mass in the left neck continued to grow and reached the size of a plum. It was pulsatile, firm, movable horizontally and unassociated with a bruit. Finger compression of the carotid artery or mass did not affect the patient adversely. Skull and chest films were normal. EKG was normal. EEG was borderline. Blood serology was positive (2+). Carotid angiography revealed a large vascular mass separating the internal and external carotid arteries and extending up to base of the skull. The angiogram was considered to be typical for a carotid chemodectoma. Operation was deferred (Figs 3-4).

Case 8. While wrestling with his young son this 51 year old man became aware of local pain in the right neck which persisted several hours. He had been aware of a painless firm



Fig 8 Aneurysm of the internal carotid artery which presented as a mass in the nasopharynx. Calcified margin of the aneurysm seen in (a)

mass in the right upper neck for several years. He had also known of gout for several years. Examination was objectively unremarkable except for the firm right cervical mass, freely movable and approximately 3 cm in diameter. Films of the skull were normal. Cervical spine films disclosed arthritic changes with marked narrowing of C6—7 interspace and straightening of cervical spine. EEG was borderline with mild left occipital dysfunction. Right common carotid angiography disclosed a tumor stain at the bifurcation of the common carotid artery with separation of the internal and external carotid arteries without narrowing of their lumina. The appearance was considered characteristic of a carotid chemodectoma. Operation was deferred (Figs 3, 5).

Case 9. On a routine examination one year previously this 23 year-old woman was found to have a small firm mass in the left neck. Two biopsies were made elsewhere and a diagnosis of angiosarcoma made following which she received 11,000 R (Cobalt) to the neck with reduction in size of the mass. Examination on admission revealed a hoarse voice, dysphagia of mild grade and a firm mass in the left neck where the skin and subcutaneous tissue were tough and fibrous in the area that had been irradiated. Skull and chest films and EEG were normal. A left carotid angiography disclosed a vascular mass approximately 7 × 4 cm in diameter at the carotid bifurcation extending up to base of skull. At operation a massive tumor extending into the left nasopharyngeal region was excised. Histologic report was chemodectoma.



Fig 7 Case 10 Chemodectomy of the right carotid bifurcation and of the right glomus jugulare. Patient had a chemodectomy of the left carotid bifurcation removed 9 years previously

encapsulated mass approximately 6×4 cm which extended superiorly and medially producing bulging of right lateral pharyngeal wall was excised. Histologic examination reported chemodectoma (Fig 3).

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Case 8 While wrestling with his young son this 51 year old man became aware of local pain in the right neck which persisted several hours. He had been aware of a painless firm



Fig. 9 Cervical neurofibroma. Catheter introduced in retrograde direction down the carotid artery. Displacement of the carotid artery in a smooth curve. Absence of tumor vessel.

Differential diagnosis

Laterally situated single mobile tumors of the neck include branchial cysts, carotid chemodectomas, neurofibromas, and metastatic lesions from the thyroid gland (LAHEY). To these must be added aneurysms of the carotid artery, enlarged glands (tuberculosis, infection, lymphomas), parotid tumors, and giant cell arteritis (PEARSE & HINSHAW 1956).

Aneurysms. Aneurysms of the carotid artery in the neck are rare when compared with aneurysms elsewhere and when present may mimic a carotid chemodectoma. Extracranial aneurysms of the carotid artery have been reported by HARDIN (1961), HALASZ & KENNEDY (1964), SPERLING & VIEHWEGER (1963), WYCHULIS, BEAHRS & BERNATZ (1964), BURTON, STEVENSON & STALLWORTH (1964). BYRNE (1958) reported a case of a mass in the neck which was wrapped with nylon mesh in the mistaken diagnosis of an aneurysm. The mass later increased in size and a carotid chemodectoma was found to be present.

WITZEL (1957) reported a case which was thought to be an aneurysm but which subsequently was found to be a carotid chemodectoma. MONRO (1950)

Case 10 This 34 year old woman had a left submandibular mass removed nine years previously, at which time it was reported to be a carotid chemodectoma. Over the next three year period an infiltrating right glomus jugulare chemodectoma was twice resected subtotally. She was readmitted to hospital because of the new finding of a growing mass in the right upper anterior part of the neck associated with mild local pruns and a drawing sensation in the neck. On examination a small plum size firm non tender mass was noted in the region of the right carotid bifurcation. In addition there were residuals of effects of her right glomus jugulare tumor and previous operations (right hemi atrophy of tongue hoarse voice, deviation of palate to right difficulty in swallowing solids diplopia on right lateral gaze and bilateral deafness — greater on right). Right carotid angiography disclosed a vascular mass in the right carotid bifurcation. At operation a 3×3 cm mass was removed from the right carotid bifurcation. Histologic report was chemodectoma (Figs 3, 7).

Case 11 This 45 year old man was asymptomatic except for the presence of a painless mass in the right neck at the level of the carotid bifurcation for six months. Examination revealed a small firm non tender mass in the region of the right carotid bifurcation which was movable horizontally but not vertically. Routine examinations including EKG EFG were normal. Right carotid angiography disclosed a vascular mass at the carotid bifurcation. This was subsequently resected and removed along with portions of the right external carotid artery. Histologic report was chemodectoma (Fig 3).

Case 12 A right carotid chemodectoma present for 10 years had been previously removed 18 years ago from a 51 year old woman. Shortly thereafter she noted a similar small painless firm mass in the left upper cervical region which grew slowly over the years to its current larger size. Occasionally while lying in bed she would perceive a regular pounding or pulsation in the mass area. She mentioned that her father also had a similar large mass in the left side on his neck which was considered to be a carotid chemodectoma by her referring physician. She stated that following the removal of her right carotid chemodectoma she had lost her voice for 8 months and that following a thyroidectomy 11 years ago she again lost her voice for 18 months. On examination a firm non tender peach sized mass was present in the left submandibular area and could be freely moved horizontally but not vertically. A bruit was audible over the tumor zone. The mass bulged slightly into the left nasopharynx. Two well healed linear surgical scars were present in the neck — a vertical one in the region of the right carotid bifurcation and a horizontal one in the suprasternal area. Routine EKG and FLC were normal. A selective left carotid angiography via an axillary catheter disclosed a large vascular mass at the bifurcation of the left common carotid artery with separation and displacement of the carotid arteries. The angiographic appearance was considered to be characteristic of carotid chemodectoma. Operation was deferred (Figs 3).

Case 13 This 57 year old man had visited several medical centers because of paroxysmal hypertension during the previous six years. A cause for this hypertension was not established. On occasion the possibility of a pheochromocytoma was ruled but not definitely confirmed. About 8 months before admission he noted a painless mass in the right submandibular area. Physical examination disclosed a plum size firm mass in the right pharynx with displacement of the tonsil and pillar anteromedially. A right carotid angiography revealed a highly vascularized mass in the right carotid bifurcation separating the internal and external carotid arteries and supplied by branches of the vertebral artery.

and do not demonstrate the angiographic appearance of a carotid chemodectoma. A primary growth elsewhere may suggest the diagnosis such as a single metastasis from a carcinoma of the thyroid gland.

Parotid tumors These occur usually in the third or fourth decade and may take years to develop. They are usually firm, hard and painless. Sialography may be helpful in the differential diagnosis (BECKER 1964).

Acknowledgement

We wish to thank Dr. John J. Conley, Chief of Head & Neck Service, St. Vincent's Hospital & Medical Center of New York, who referred many of the patients including all those with chemodectomas for diagnostic evaluation and angiography.

SUMMARY

The clinical and angiographic features associated with chemodectomas of the carotid bifurcation have been reviewed and our experiences including 13 patients with this disease are presented. The usefulness and role of angiography in the differential diagnosis of cervical masses in the region of the carotid bifurcation is demonstrated.

ZUSAMMENFASSUNG

Die Verfasser haben eine Übersicht über die klinischen und angiographischen Symptome der mit Chemodectom der Karotisbifurkation verbunden sind gemacht. Ihre Erfahrungen einschliesslich 13 Patienten mit dieser Krankheit werden vorgelegt. Der Wert und die Rolle der Angiographie für die Differentialdiagnose der Tumoren in Bezirk der Karotisbifurkation werden demonstriert.

RÉSUMÉ

Les auteurs ont passé en revue les signes cliniques et angiographiques des chemodectomes de la bifurcation carotidienne et présentent leurs 13 cas personnels. Ils montrent l'utilité de l'angiographie pour le diagnostic différentiel des tumeurs cervicales de la région de la bifurcation carotidienne.

BIBLIOGRAPHY

- ALBERS C. D. Branchial anomalies. *J. Amer. med. Ass.* 186 (1963) 399.
 ANDERSON R. and SCARCELLA J. V. Carotid body tumors. *Amer. J. Surg.* 106 (1963) 836.
 ANIKHON V. V. and BELENKH M. L. Pharmacology of the carotid body chemoreceptors.
 Translated by Dr. R. Crawford. p. 225. MacMillan, 1963.
 AREAN V. M. Recurrent carotid body tumor. *Arch. Path.* 60 (1955) 530.
 — and DEARELLANO C. A. R. Intra abdominal non-chromaffin paraganglioma. *Ann. Surg.* 144 (1956) 133.
 ARKINSON H. M., EPPENSTEIN S. S. and HERZBERGER E. E. Tumors of the glomus jugulare co-existing possibly with carotid body tumor. *Acta neurochir.* 3 (1953) 170.

reported a case of carotid ligation for an aneurysm which turned out to be a carotid chemodectoma. Lipschitz (1958) distinguished between a highly vascular tumor and an aneurysm. An angiography revealed a carotid chemodectoma.

Angiomas and other vascular tumors of the neck may bear some similarity to the angiographic appearance of carotid chemodectomas. These rarely displace vessels and usually present no difficulty in the clinical diagnosis of the case.

Case 7 in our series was admitted with the diagnosis of an aneurysm. His history went back many years. A gradually expanding mass in the neck was considered an aneurysm. He had a positive serology for syphilis and had many courses of treatment. Angiography revealed a carotid chemodectoma (Fig. 4). Two other cases, not included in our series but admitted with the diagnosis of carotid chemodectoma, had an aneurysm of the carotid artery and a tortuous vessel respectively. A carotid angiography made the distinction immediately. One case admitted to us with a mass in the neck had calcification in the wall of the aneurysm (Fig. 8).

Branchial cysts These may occur at the level of the carotid bifurcation and may be mistaken for carotid chemodectomas. They are inclined to be more superficial and are usually below the level of the carotid bifurcation. They rarely bulge into the pharynx and can be moved in a horizontal and vertical plane. A detailed history, including embryonic developmental abnormalities, a barium swallow with contrast medium and cinefluoroscopy will usually help make the diagnosis (ALBERS 1963).

Neurofibroma These may arise from any of the cervical nerves and may be extremely difficult to distinguish from carotid chemodectomas, particularly if they happen to grow between the internal and the external carotid arteries which may be separated. If they arise from a nerve which has a vertical disposition then vertical movement will be limited. These tumors are usually immobile and enlarge the exit foramina of the spine. Evidence of masses elsewhere should be sought as well as the other stigmata of neurofibromatosis. Although neurofibromas may be associated with increased vascularity, the angiographic appearance in no way resembles a carotid chemodectoma (Fig. 9).

Lymph nodes Neoplastic or infected nodes may sometimes cause difficulty in the differential diagnosis. Calcification may be seen in tuberculous glands. Lymph nodes rarely separate the internal and the external carotid arteries

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BIBLIOGRAPHY

- ALBERS C. D. Branchial anomalies. *J. Amer. med. Ass.* 186 (1963) 399.
 ANDERSON R. and SCARCELLA J. V. Carotid body tumors. *Amer. J. Surg.* 106 (1963) 856.
 AVCHUKOV S. V. and BELENKIN M. L. Pharmacology of the carotid body chemoreceptors. Translated by Dr. R. Crawford. p. 295. MacMillan, 1963.
 ARON V. M. Recurrent carotid body tumor. *Arch. Path.* 60 (1955) 530.
 — and DEANELLANO G. A. R. Intra-abdominal non-chromaffin paraganglioma. *Ann. Surg.* 144 (1956) 13.
 KRENN H. M., EPPENSTEIN S. S. and HERZBERGER E. E. Tumors of the glomus jugulare co-existing possibly with carotid body tumor. *Acta neurochir.* 3 (1963) 170.

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- HEYLMAN C, BOLCKAERT J J et DAUTREBANDE L Sinus carotidien et reflexes respiratoires Arch int Pharmacodyn 39 (1930) 400
- IDDOHRY H Angiographic diagnosis of carotid body tumors Acta radiol 30 (1901) 110
- KIPKE G F Simultaneous chromaffin tumors of the carotid body and the glomus jugulare Arch Path 44 (1947) 113
- KROLL A J, ALEXANDER B, COCHIOS F and PECRET L Hereditary deficiencies of clotting factors VII and X associated with carotid body tumors New Engl J Med 278 (1964) 6
- LADENHEIM J C and SAGHS E JR Familial tumors of the glomus jugulare Neurology 11 (1961) 303
- LAHEY F H and WARREN K W Tumors of the carotid body Surg Gynec Obstet 80 (1947) 281
- — Long term appraisal of carotid body tumors with remarks on their removal Surg Gynec Obstet 92 (1901) 481
- LATTEI R Non-chromaffin paraganglioma of ganglion nodosum carotid body and aortic arch bodies Cancer 3 (1950) 667
- MacDONALD J J and SPROLL E Non-chromaffin paraganglioma of carotid body and orbit report of a case Ann Surg 139 (1954) 382
- LECOMTE P M Tumors of the carotid body and related structures (chemoreceptor system) Atlas of Tumor Pathology Armed Forces Institute of Pathology Wash DC 1901
- LEWISON E F and WEINBERG T Carotid body tumors Surgery 27 (1950) 437
- LEHTEHAUER F Operationsindikation und Arteriographie bei Carotidruessengeschwulsten Zbl Chir 65 (1938) 2286
- LIPSCHITZ R Angiographic study of a carotid body tumor Brit J Radiol 31 (1908) 105
- MACCONE W S Carotid body tumors Ann Surg 127 (1948) 269
- MACDONALD R A A carotid body like tumor on the left subclavian artery Arch Path 62 (1906) 107
- MARCHAND F Beitrage zur kenniniss der normalen und pathologischen anatomic der glandula carotica Festschr f Virchow (1891) 1535
- MARTORELL F Tumor of the carotid body Angiology 7 (1906) 278
- MELKRATH D C and REMINE W H Carotid body tumors Surg Clin N Amer 43 (1963) 1130
- MIRON A C Benign and malignant carotid body tumors J Amer med Ass 181 (1962) 13
- MOBERG A Malignant carotid body tumor with metastasis in lungs Acta otolaryng 53 (1961) 500
- MOYER R S The natural history of carotid body tumors and their diagnosis and treatment with a report of five cases Brit J Surg 37 (1950) 445
- MURPHY H M, SWAN H and TAYLOR E R Carotid body tumors Report of twelve cases including one case with proved visceral dissemination AMA Arch Surg 67 (1903) 114
- MORRIS C C JR, BALAS P E, COOLEY D A, CRAWFORD E S and DEBAKEY M E Surgical treatment of benign and malignant carotid body tumors Amer Surg 29 (1963) 429
- MULLER AN R M Chemodectoma in the dog Amer J Path 26 (1900) 680
- MURPHY H J A and BERK M E Carotid body tumors S Afr med J 33 (1909) 329
- NELSON W R Carotid body tumors Surgery 51 (1967) 326
- ODERFELTZER R J H and TOFANI W C The Handbook of Physiology Vol 2 p 1123 Williams & Wilkins Baltimore 1960
- PIARRY H F and HINSLEY J P Bilateral arteritis simulating carotid body tumors Surg Gynec Obstet 103 1906 263

- BECKER W Parotid tumors, diagnosis differential diagnosis therapy Physicians Panorama 2 (1964) 25
- BIRRI LL J H W Carotid body tumors a study of three cases including a bilateral example Aust N Z J Surg 22 (1952) 123
- BORELLI G Aspetti angiografici dei tumori del glomo carotideo Radiol med 45 (1959) 250
- BOYD J D The development of the human carotid body Contributions to Embryology Vol 26, p 3 Carnegie Institute 1937
- BURMAN S O The vagal body tumor Ann Surg 141 (1955), 488
- The chemoreceptor system and its tumor — the chemodectoma Int Abstr Surg 109 (1956) 330
- BUXTON J I STEVENSON T B and STALLWORTH J M Arteriosclerotic aneurysm of the extracranial internal carotid artery treated by excision and primary reanastomosis under controlled hypertension Ann Surg 159 (1964) 222
- BYRNE J J Carotid body and allied tumors Amer J Surg 95 (1958) 371
- CHAST W H Unilateral and bilateral tumors of the carotid body J Path Bact 36 (1933) 1
- CONLEY J J The management of carotid body tumors Surg Gynec Obstet 12 (1963) 722
- CHUSID J C and SCHECHTER M M Angiography in head and neck surgery Arch Surg 89 (1964) 609
- DESAI M G and PATEL C C Heredo familial carotid body tumors Clin Radiol 12 (1961) 21
- DONALD R A and CRILE G Tumors of the carotid body Amer J Surg 75 (1948) 433
- DUNCAN D K and McDONALD J R Chemodectoma of the mediastinum two cases from right posterior costovertebral sulcus Am J clin Path 21 (1951) 515
- Editor Biography of a word J Amer med Ass 187 (1964) 1021
- ENGSTROM H and HANBLERGER C A Bilateral tumor of the carotid body Acta otolaryng 48 (1957) 390
- FANNING J I Woods F M and CHRISTIAN H J Metastatic carotid body tumor J Amer med Ass 185 (1963) 129
- FERNAN ZECARPA L Revista di patologia e clinica chirurgica 2 (1957) 171
- ISHIER F R and HAZARD J B Non chromaffin paraganglioma of the orbit Cancer 5 (1959) 521
- FRIDMAN E N and LAU R Carotid body tumors J Mt Sinai Hosp 24 (1957) 633
- GANONG W F Review of Medical Physiology P 553 Lance Medical Publications 1963
- GILFORD H and DAVIS K L H Isolated tumors of neck and their origin as endotheliomata of carotid body with account of three cases Practitioner 73 (1904) 729
- GOARMAGIICH N and PATTYN S A presumably benign tumor and a proved malignant tumor of the carotid body Amer J Path 30 (1934) 679
- GOODOR I I and LISCHER C L Tumor of carotid body and pancreas Arch Path 35 (1943) 906
- GRONBERGER G Sarcoma of carotid gland Hygiea 79 (1917) 169
- HALASZ N A and KENNEDY J C Excision of arteriosclerotic aneurysms of the cervical internal carotid artery J Neurosurg 21 (1964) 352
- HARDIN C A Carotid body tumors and aneurysms Angiology 12 (1961) 597
- HARRINGTON S W CLAGETT O T and DOCKERTY M H Tumors of the carotid body Clinical and pathologic considerations of twenty tumors affecting 19 patients (one bilateral) Ann Surg 114 (1941) 820
- HAWKINS T D Glomus jugulare and carotid body tumors Clin Radiol 12 (1961) 199

MORPHOLOGICAL CHANGES OF THE BRAIN FOLLOWING INTRATHECAL APPLICATION OF CONTRAST MEDIA

by

REINHARD SCHÖBER

Positive contrast media are still used in many countries for ventriculography as well as for myelography. Numerous reports of complications have, however, been published (adhesive arachnoiditis, brain nerve damage, blindness (3, 6, 9, 12)). When iodized oil was succeeded by iodized esters like Pantopaque, such complications did not disappear in examinations of the spinal and intracranial spaces (2, 5, 10, 13, 14). In spite of all efforts the contrast medium can never be completely removed. In ventriculography it can not be prevented from reaching the cisterns unless there is a complete occlusion. This is even more likely to happen in myeloencephalography with Pantopaque as MOVES & WERMAN (1959) have pointed out: here the removal of the contrast medium is absolutely impossible.

It seems rather strange, under these circumstances, that quite recently publications from Latin countries have reported on 1 280 and 3 000 ventriculographies using iodized oil (MORETE de PARDAL & PARDAL 1958, DILLENCE et coll. 1961). In Germany and the Scandinavian countries its use has been

- PETTET J R, WOOLNER L B and JUDS E S Carotid body tumors (chemodectomas) *Ann Surg* 157 (1953), 465
- PHELPS F W, CASE S W and SNYDER G A C Primary tumors of carotid body review of 159 histologically verified cases, report of a case *West J Surg* 45 (1937) 42
- RABSON A S and ELLIOT J L Carotid body tumors with regional lymph node involvement with report of a case *Surgery* 42 (1957) 381
- RANDALL K J and WALTER J B Metastasizing non chromaffin paraganglioma of thigh *J Path Bact* 67 (1954), 69
- RUEST H F, LUCAS R N and BERGMAN P A Malignant carotid body tumors *Ann Surg* 157 (1963) 232
- RIEGER Cited by Byrne J J (1958) 377
- ROMANSKI R Chemodectoma (non chromaffin paraganglioma) of carotid body with distant metastases *Amer J Path* 30 (1954), 1
- ROSS I L Electron microscopic observations of the carotid body of the cat *Biophys Biochem & Cytol* 6 (1959) 253
- RUSSE B I Current concepts on the treatment of carotid body tumors *Surgery* 52 (1962) 679
- SCHADE R Tumors of the glomus jugulare and glomus caroticum *Brit J Cancer* 7 (1953) 440
- SESSIONS R T, McSWAIN II, CARLSON R I and SCOTT H Surgical experiences with tumors of the carotid body glomus jugulare and retroperitoneal non chromaffin paraganglia *Ann Surg* 150 (1959) 808
- SHOWAN H K and OWEN C I Carotid body tumors *Amer J Surg* 40 (1938) 462
- SHETANA H F and SCOTT W F JR Malignant tumors of non chromaffin paraganglia *Milit Med* 109 (1951) 330
- SPEERLING M and VIEHWEGER G Extracranial aneurysms of the internal carotid artery and their treatment *Chirurg* 34 (1963), 369
- SPOTNITZ M Case of malignant tumor of carotid body with multiple metastases *Oncologia* 4 (1951) 239
- SUTTON D Angiography Livingstone 1962
- TAYLOR G G On carotid tumors *Brit J Surg* 28 (1940) 163
- TURNBULL I M JR Malignancy in carotid body tumors with presentation of a proved case of metastasis to the lungs *West J Surg* 62 (1964) 382
- UNNAS B Central cardiovascular control *Handbook of Physiology* Vol 2 p 1131 Williams & Wilkins Baltimore Md 1960
- VON HALLER Cited by DICKINSON A M and GRAVER C A Carotid body tumors Review of the literature with a report of 2 cases *Amer J Surg* 69 (1944) 9
- WARREN K W Some observations on carotid body tumors *Surg Clin N Amer* 39 (1950) 621
- WESTBURY G The management of carotid body tumors with a report of 7 cases *Brit J Surg* 47 (1960) 605
- WITZEL N Carotid angiography in diagnosis and treatment of tumors of the neck *AMA Arch Surg* 74 (1957) 954
- WOSNESSENKI Cited by TAYLOR G G (1940)
- WRIGHT I B A case of carotid body tumor investigated by angiography *J Fac Radiol* 3 (1952), 290
- WYCHIULIS A R, BEAVERS O H and BERNATZ P F Aneurysm of internal carotid artery treated by excision and anastomosis to external carotid artery *AMA Arch Surg* 88 (1964) 803

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Fig 1



Fig 2

Fig 1 Position of the contrast medium in the basal cisterns

Fig 2 Eighteen weeks after injection. On the removed brain the contrast medium is partly fixed to the leptomeninges

restricted, while it is more frequently used in the United States and Great Britain

Although there have been numerous clinical reports of complications and results of autopsies or biopsies of pathological changes after the use of Pantopaque within the cerebrospinal fluid spaces, no experimental work has been done. The authors therefore started experiments on rabbits.

Three possibilities for the application of the contrast medium were at hand

1 Puncture of the great cistern, with this technique it is difficult to pass the contrast medium into the skull cavity without its flowing off into the spinal canal

2 Puncture of the ventricular system: this has the disadvantage of unavoidable traumatic brain damage and hence a changed situation for the assessment of the lesions

3 Trephination of the skull cap in the frontal area with a ball drill and injection of the contrast medium through a tiny perforation of the dura into the subarachnoid space of the convexity. The last mentioned method was used, since with this technique trauma is avoided

An amount of 0.1 ml/kg was injected, that is, 0.3 ml on an average per animal. Roentgen ray control showed that the contrast medium flowed quickly down into the area of the basal cisterns and remained there (Fig 1)

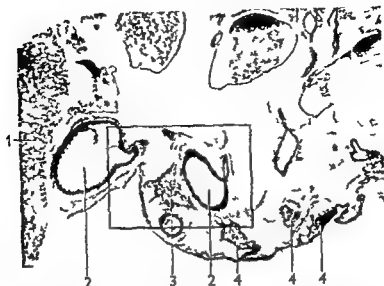


Fig 3 5 weeks after injection. Densification of the arachnoidal meshwork with plaques in the cisterna chiasmatis. 1 Tractus opticus 2 Carotis interna 3 Pia mater on carotid 4 Arachnoidal cell cones. Hematoxylin-eosin $\times 47$

Thus the same situation as in ventriculography and myelography was created.

The animals were under observation for up to 4 1/2 months. There were no side effects. The contrast medium did not change position or extension during this period and later roentgen control only showed the droplets in closer formation, a sign of sealing-off.

The removal of the brain at weekly intervals was in some cases not so easy as in untreated animals. After the brain had been carefully lifted from the anterior fossa adhesions were to be seen even macroscopically. Even the dura, normally fixed to the bone, came away. Pantopaque flowed out from the opened cisterns. Radiograms of the removed and fixed brain showed traces of contrast medium within the basal cisterns, apparently adherent to the meningeal tissue (Fig. 2).

In all cases the histological examination revealed the same findings, although they differed in intensity according to the duration of the test. The arachnoidea consisted of a visceral endothelial layer connected with the pia and a parietal layer in connection with the dura. A filamentous meshwork extended between them. Bedlike proliferation areas had developed on these endothelial layers which amalgamated to form cell cones. Undoubtedly the perivascular tissue of the neighbouring pia vessels had taken part in developing these cell cones. They were a formation of rather large epithelial cells which both

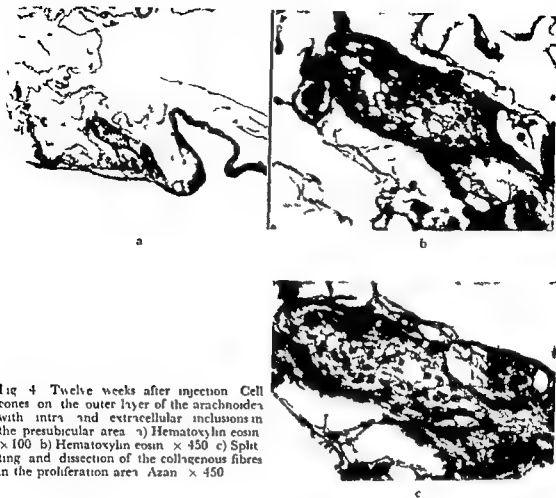


Fig 4 Twelve weeks after injection Cell cones on the outer layer of the arachnoides with intra and extracellular inclusions in the presubicular area a) Hematoxylin eosin $\times 100$ b) Hematoxylin eosin $\times 450$ c) Splitting and dissection of the collagenous fibres in the proliferation area Azan $\times 450$

intracellularly and extracellularly had some small droplike inclusions. Some times large cavities within the meshwork could be seen — undoubtedly the places from which the oily contrast medium was resolved during the preparation of the tissue slides (Figs 3 and 4).

For the demonstration of the collagenous fibres Azan dye was used. They were found to be dissected and fragmented within the area of the cell plaques and cell cones. An increase in the collagenous fibres and densification of the arachnoidal meshwork were also seen (Figs 4c and 5).

It should be stressed that the changes observed were not inflamed granulation tissue, they completely lacked participation by the capillaries. They were a proliferative mesenchymal reaction with intra- and extracellular inclusions, fibre dissection and fibre increase. It was an adhesive process. As reported in some cases in humans, genuine granulation tumors apparently develop under special conditions. They depend on the consistence of the cerebrospinal fluid,



Fig. 5 Eighteen weeks after injection. Arachnoidal proliferation area in the cisterna chiasmatis showing increase in collagenous fiber concentration and splitting of the collagenous fibres. Azan $\times 100$.

admixture of blood and so on (2, 13). None of these circumstances were present in our experiment.

The initial damage is caused by slow decomposition of the iodine esters liberating free fatty acids here the undecyl acid which saponifies with the potassium of the CSF. This causes a chronic irritation. It is difficult to say how far the iodine separated from the molecule could also be responsible for these reactions. In any case it can only cause an effect in elementary form and not as an ion. The diminishing of the Pantopaque depots which is said to take place in humans at a rate of 1 ml per year is due according to several statements (HURTEAU et coll 1954) to iodine elimination and not to true resorption of the carrier molecule which remains in the same place.

According to the results of the experiment the use of iodine esters within the cerebrospinal fluid spaces is not without risk. Whether or not the arachnoid adhesions become efficient from the clinical point of view depends solely on the localisation of the rest depot of the contrast medium and therefore is absolutely accidental. The risks are probably less in the spinal area than in the vicinity of the brain especially of the cranial nerves where such adhesive changes may lead to serious dysfunction. Consequently, the use of substances that are neither resorbable nor removable is inadvisable. As long as there is not an absolutely indifferent contrast medium for the examination of these

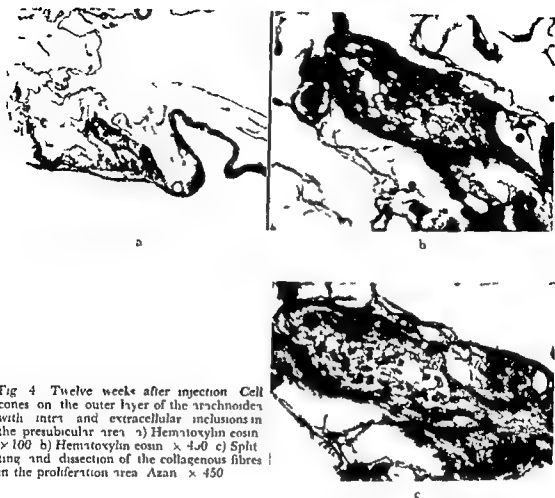


Fig 4 Twelve weeks after injection. Cell cones on the outer layer of the arachnoid with intra and extracellular inclusions in the presubicular area. a) Hematoxylin-eosin $\times 100$ b) Hematoxylin-eosin $\times 450$ c) Splitting and dissection of the collagenous fibres in the proliferation area Azan $\times 450$

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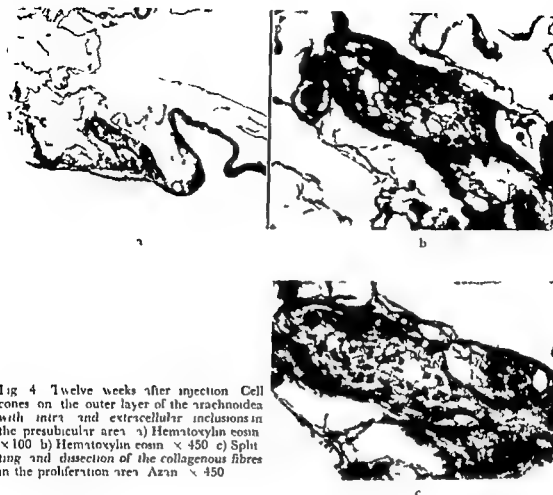


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intracerebral vessels seemed noticeable at this time. This fact may be due to an increased exchange of substance and fluid between brain tissue and cerebrospinal fluid space or blood vessels.

Although during these seizures a specially high demand for energy is being put upon the brain, there is no damage to the producers of the energy connected with it. Damage as such will only be caused by cerebral hypoxemia due to disregulation of breathing and of the circulatory system during the seizures. Under general anesthesia, brain damage due to cerebral hypoxemia may be shown above, be avoided.

SUMMARY

Experiments on rabbits after injection of iodine esters in the intracranial fluid spaces showed after several weeks proliferative mesenchymal reactions of the arachnoidea with intra and extracellular inclusions and dissection and increase in fibres. Their use for diagnostic purposes is therefore not without risks. The resorption of water soluble contrast media does not cause morphological changes in the brain. The practical use of these substances is reduced since they cause seizures that can only be avoided by general anesthesia.

ZUSAMMENFASSUNG

Injektion von Jodestern in die intracranialen Liquorraume von Kaninchen bestätigte nach einigen Wochen das Auftreten von proliferativen mesenchymalen Reaktionen der Arachnoidea mit intra und extrazellulären Einschlüssen, Zerteilung und Zunahme der Fasern. Ihre Anwendung für diagnostische Zwecke ist deshalb nicht ohne Risiko. Die Resorption von wasserlöslichen Kontrastmitteln verursacht keine morphologischen Gehirnveränderungen. Die praktische Anwendung dieser Substanzen für diagnostische Zwecke ist eingeschränkt, da sie Krämpfe verursachen, die nur mit Allgemeinanästhesie verhindert werden können.

RÉSUMÉ

L'injection expérimentale chez des lapins d'esters iodés dans les espaces liquidiens intracrâniens a été suivie au bout de plusieurs semaines de réactions prolifératives du mésenchyme de l'arachnoïde avec inclusions intra et extracellulaires, dissection et augmentation du nombre des fibres. Leur emploi pour le diagnostic n'est donc pas sans danger. La résorption des moyens de contraste hydrosolubles ne cause pas de modifications morphologiques du cerveau. L'utilisation pratique de ces substances est réduite car elles causent des crises convulsives qu'on ne peut éviter que par anesthésie générale.

REFERENCES

1. DILLON D., DAVID M. und TALAIRACH J. Zur Indikation und Technik der Jodventriculographie. *Neurochirurgie* 6 (1961) 347.
2. ERICSON T. C. and van BAAREN H. J. Late meningeal reaction to ethyl iodophenyl undecylate used in myelography. *J. Amer. med. Ass.* 153 (1953) 636.

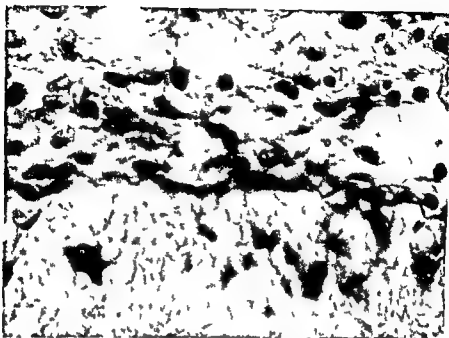


Fig. 6 Two days after injection of water soluble contrast medium. Widening of the subpial glia chambers with cell infiltrates in pia and arachnoidea (precentralis). Hematoxylin eosin $\times 450$.

sensitive areas we must rely on air, which in most cases gives enough information to the skilled examiner.

Further examinations were performed with resorbable contrast media (diatrizoate). After the injection of 0.12 ml/kg (Urografin 60%) into the fluid space the animals reacted with spasms and died. On the other hand, a 5% NaCl solution was well tolerated. Thus, hypertonicity, which is higher in the NaCl solution, was not the important factor in causing the spasms.

After injection of the contrast medium under general anesthesia no unusual reactions were observed. The anesthesia extends the time of resorption of the contrast medium to at least 2 hours, however. The spasm producing tendency of the solution may be lessened in this way, and the amount can even be doubled without damage.

These brains were examined histologically at intervals of up to 4 weeks after injection. They revealed neither damage of the ganglion cells nor progressive or regressive reactions of the glia that could indicate perishing of neurons. Only during the first days was loosening of the fine reticular tissue of the pia and cell infiltrates seen (Fig. 6). However, these lesions were quickly reversible and were also seen in animals that received Ringer's solution alone.

Widening of the subpial glia chambers and periaxonal spaces of the

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REFERENCES

1. DIEHLG, D., DAVID, M. and TALAMRACH, J. Zur Indikation und Technik der Jodventrilo-graphie. *Neuro-chirurgie* 6 (1961) 347.
2. ERICKSON, T. C. and van BAAREN, H. J. Late meningeal reaction to ethyl iodophenyl undecylate used in myelography. *J. Amer. med. Ass.* 153 (1953) 636.

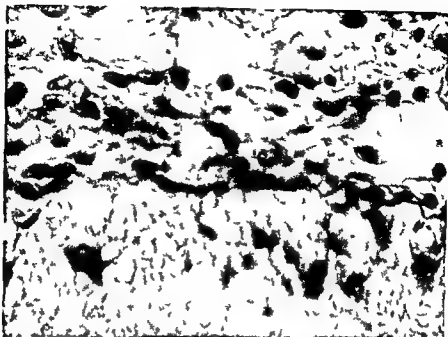


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SAFER CAROTID ANGIOGRAPHY BY PERCUTANEOUS CANNULATION

by

PHILIP SHELTON

The greatest danger of carotid angiography lies in the possibility of dissection of the wall of the artery by the point of the needle thus obstructing or reducing the intracranial blood flow (Fig 1) SALTZMAN suggested that this complication occurred in 10 % of patients examined there was no reason to suppose that our experience with needle angiography differed from his SCHEINBERG & ZUNER (1963) found intramural injections in 60 % of patients undergoing carotid angiography who later exhibited complications which in their series of 500 patients arose in 3.7 % of examinations and included 3 deaths Catheterisation (LIVERUP 1958 ELVIN 1960 AMUNDSEN, DIETRICHSEN, ENGE & WILLIAMSON 1963 and SCHECHTER 1963) and teflon cannulation (SOILA 1963) are alternative methods which have been suggested to avoid this danger The purpose of this paper is to describe another technique which we think is far easier to perform than catheterisation or teflon cannulation greatly increases the safety of the operation compared with needle angiography and permits selective injection of the internal or external carotid arteries almost at will

The instrument (Fig 2) which has been in use since October 1962, is a short

- 3 HOFMANN A Die Bedeutung der Röntgendiagnostik für die operative Behandlung des Bandscheibenprolapses Fortschr Röntgenstr 73 (1950), 412
- 4 HURTTAU E F BAIRD W C and SINCLAIR C Arachnoiditis following the use of iodized oil J Bone Jt Surg 36A (1954), 393
- 5 LUCH I C LEITH W and BURRACE W S Pantopaque meningitis due to sensitivity Radiology 57 (1951), 878
- 6 MIRKA P Über Komplikationen der Myelographie Wien klin Wschr (1949), 700
- 7 MONES R and WERMAN R Pantopaque myelocencephalography Radiology 72 (1959) 803
- 8 MORITZ de PARDAL M L et PARDAL F Mise au point de l'iodoventriculographie Acta radiol 50 (1958), 34
- 9 PANTER K Die Kontrastmitteldarstellung der spinalen Räume Fortschr Neurol Psychiat 23 (1955) 173
- 10 PRACHAR W G and ROBERTSON R C L Pantopaque myelography Results comparison of contrast media and spinal fluid reaction J Neurosurg 2 (1945) 220
- 11 RALSTON B L GROSS S W and NEWMANN E W Pantopaque ventriculography in the localization of surgical lesions of the posterior fossa Amer J Roentgenol 81 (1959) 972
- 12 RODRIGUEZ de MATA T Un caso de amaurosis total y transitoria consecutiva a una inyección intrarraquídea de lipiodol Acta Soc cir Madrid 2 (1933) 165
- 13 TARLOW I M Pantopaque meningitis disclosed at operation J Amer med Ass 129 (1945) 1014
- 14 WINNLMANN N W GOTTEN N and SCHULBERT D Localised adhesive arachnoiditis Study of 25 cases with reference to etiology Trans Amer neurol Ass 78 (1953) 15

SAFER CAROTID ANGIOGRAPHY BY PERCUTANEOUS CANNULATION

by

PHILIP SHELDON

The greatest danger of carotid angiography lies in the possibility of dissection of the wall of the artery by the point of the needle thus obstructing or reducing the intracranial blood flow (Fig 1) SALTZMAN suggested that this complication occurred in 10 % of patients examined there was no reason to suppose that our experience with needle angiography differed from his SCHEINBERG & ZUCKER (1963) found intramural injections in 60 % of patients undergoing carotid angiography who later exhibited complications which in their series of 500 patients arose in 3.7 % of examinations and included 3 deaths Catheterisation (LIVERUD 1958 ELVIN 1960 AMUNDSEN DIETRICHSEN FAGE & WILLIAMSON 1963 and SCHECHTER 1963) and teflon cannulation (SOILA 1963) are alternative methods which have been suggested to avoid this danger The purpose of this paper is to describe another technique which we think is far easier to perform than catheterisation or teflon cannulation greatly increases the safety of the operation compared with needle angiography and permits selective injection of the internal or external carotid arteries almost at will

The instrument (Fig 2) which has been in use since October 1962 is a short



Fig 1 Angiograms showing almost complete obstruction by a small intramural deposit of contrast material during angiography with a conventional needle a) Arterial phase b) Two seconds later c) Arterial phase a few minutes later after puncture lower in the neck (Courtesy Brit J Radiol)



Fig 2 From above Trochar cannula and guide wire held by adjustable chuck (Courtesy, Brit J Radiol)

thin walled cannula made of 18 SWG metal tubing containing a sharp trochar. Inside diameter is greater than 0.95 mm, and thus accommodates a Seldinger 160 guide wire (SELDINGER 1953). The guide wire we use is 15 cm long and is held by an adjustable chuck (SHILDON 1964).

The artery is punctured in the usual way by transfixing both walls and with drawing the cannula after removal of the trochar. Since we are usually anxious to obtain films of the internal carotid artery distribution without overlying confusing external carotid branches and dilution of contrast (INDGREN 1947,



Fig. 3 Selective cannulation of external carotid artery. Even though the origin of the external carotid artery does not appear percutaneous cannulation was easily carried out.

WICKBOM 1948) we find it best to make the puncture at the bifurcation of the common carotid artery. The cannula may then be passed into the internal or external arteries (Fig. 3) after a small preliminary injection to exclude stenosis or atheromatous roughening and to show how the vessels lie. Cannulation is done by inserting the guide wire through the cannula so that its flexible tip projects by 2 or 3 cm. and then either sliding the cannula up the guide wire or passing the two up together. The cannula should not be passed without the guide wire in position since abrasion or other damage to the wall may be caused.

It is obvious that the guide wire must be handled delicately and with care, its use is better avoided if a preliminary injection shows roughening of the arterial wall. Some objection may be raised that it may be dangerous to pass a guide wire in the region of the carotid bifurcation, but in only one patient has there been a neurological complication which could be ascribed to use of the guide wire and in this case an obvious contraindication in the form of a large cauliflower like plaque on the posterior arterial wall was ignored. This underlines the necessity for obtaining a film of the region of the carotid bifurcation especially in elderly patients.



Fig. 4 Tip of cannula is hard against posterior wall. Even in this poor position no dissection has occurred which a needle would almost certainly have caused. A puncture nearer the bifurcation would have been more satisfactory. (Courtesy Brit J Radiol)

While internal carotid puncture with a needle may often be a difficult and tedious procedure, with the cannula the internal carotid artery has been cannulated in virtually every case during at least the past eighteen months unless contra-indicated. NEW & BAKER (1963) recently described a similar cannula, but theirs does not seem to allow any facility for easy and safe selective cannulation.

Our cannula has been in use in more than 3 000 examinations. Evidence of injection of contrast material into the arterial wall has fallen, so that this occurrence is now only seen once in every 150 to 200 angiograms. Similarly the number of complications recorded in our patients is extremely low compared with many published series, due, we believe to lack of damage to the vessel wall (although even when needle angiography used to be carried out we were never greatly troubled by complications). Other advantages over the conventional needle are that the head may be moved with impunity for various projections, and adjustments to the cannula are very rarely needed (Fig. 4) while beginners learn the technique as easily as with a needle, but with far greater safety for the patient.

While the polythene catheter must obviously be more versatile than a cannula the cannula has the advantage of simplicity, and has small dimensions, since it is only 1.20 mm in diameter, compared with 1.60 mm of the PE 160 polythene catheter. Only one manoeuvre is needed to insert it in the artery, and there is no need to replace the cannula by a blunt polythene catheter

which must cause additional trauma to the arterial wall. Dissection can also occur with polythene catheterisation. The technique is no more difficult than conventional needling which is another great advantage compared with catheterisation.

Compared with teflon cannulation the cannula which the present author uses has the advantage that it cannot kink or corrugate when passed through the skin or vessel wall. In addition a teflon cannula being easily bent often cannot be directed for selective cannulation.

Acknowledgements

I am grateful to Dr F. H. Kemp for his advice and help in the preparation of this paper. The instruments described are made for the author by V. J. Millard Ltd, 36 Highgate Hill, London N. 19.

SUMMARY

A small but useful contrivance for angiography has been described. It is as simple to use as more versatile and far safer than a needle. In the interests of the patients' safety and comfort during carotid angiography it is considered that use of the needle should be discontinued.

ZUSAMMENFASSUNG

Es wird eine kleine, jedoch nützliche Anordnung für Carotisangiographie beschrieben. Sie ist einfach in der Anwendung, vielseitiger und bei weitem sicherer als eine Nadel, die im Interesse der Sicherheit und Bequemlichkeit für den Patienten nicht mehr angewendet werden sollte.

RÉSUMÉ

L'auteur décrit un petit perfectionnement instrumental pour l'angiographie d'emploi simple et beaucoup moins dangereux qu'une aiguille. Il pense que pour la sécurité et le confort du malade pendant l'angiographie on devrait abandonner l'emploi de l'aiguille.

REFERENCES

- WILKINSON P., DIETRICHSEN P., ENGE I. and WILLIAMSON R. Cerebral angiography by catheterization. *Acta radiol.* 10 (1963) 164.
ELVIN P. Angiography of the carotid artery with catheter technique. *Radiology* 75 (1960) 80.
LINDGREN E. Technique of direct (percutaneous) cerebral angiography. *Brit. J. Radiol.* 75 (1947) 376.
LIVERLID K. Occluding dissecting aneurysm as a complication of carotid angiography. *J. Oslo City Hospital* 8 (1958) 209.
NEW P. F. J. and BAKER E. Technique for arterial puncture. *J. Neurosurg.* 20 (1963) 390.



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VESSEL CALIBER AND CIRCULATION TIME AT CEREBRAL ANGIOGRAPHY IN MONKEYS UNDERGOING HYPOTHERMIA

by

M C SMITH T H NEWTON and R PEARSON

The reaction of cerebral vessels to hypothermia has been obscured under both clinical and experimental conditions by factors of viscosity and ventilation (1 2 3 4) The viscosity of blood is increased as it is cooled in the course of hypothermia resulting in an increased resistance to flow Because of the lowered metabolic rate respiration continued at normal rates under hypothermic conditions markedly lowers the arterial pCO_2 , constricting the cerebral vessels Cerebral blood flow, therefore, may be decreased in the hypothermic state both by cerebrovascular constriction and increased blood viscosity

Our purpose in this study was to determine the effects of hypothermia per se on the caliber of the cerebral vessels and on the circulation time while viscosity and arterial pCO_2 were carefully controlled Vascular response to changes of the arterial pCO_2 in the hypothermic state was also studied The monkey was chosen for these experiments because its cerebral circulation is similar to that of man Vessel caliber and circulation time were measured using angiographic techniques

- SAITZMAN G I : Angiographic demonstration of the posterior communicating and posterior cerebral arteries. *Acta radiol* 52 (1959), 1
- SCHLEGTER M : Percutaneous carotid catheterization. *Acta radiol* 1 (1963) 417
- SCHLEINBERG P and ZUNZER F : Complications in direct percutaneous carotid angiography. *Arch Neurol* 8 (1963) 676
- SHELDON P : Catheter replacement of the needle in percutaneous arteriography. *Acta radiol* 39 (1953) 368
- SHELTON P : Percutaneous cannulation of the carotid artery. *Brit J Radiol* 37 (1964) 526
- SOHA P : Introduction of a soft cannula into the carotid artery by percutaneous puncture. *Acta radiol* 1 (1963) 451
- WICKHAM I : Cerebral angiography. *Acta radiol Suppl* 72 (1948)

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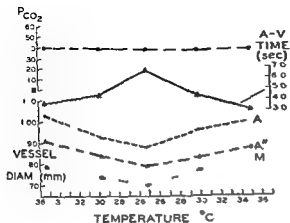


Fig 2 Effect of cooling and re warming on vessel size and circulation time during maintenance of normal pCO_2

an automatic injector. At intervals of 0.7 sec 10 films were obtained with a Sanchez Perez film changer. The calibers of the anterior cerebral and middle cerebral arteries were measured by projection techniques. These measurements were made in the inferior and superior portions of the pericallosal artery and in the middle cerebral artery near the trifurcation. These sites have been designated A₁, A₂, and M respectively (Fig 1). Arteriovenous circulation time was measured from filling of the carotid siphon to filling of the internal cerebral vein. The experimental animals were divided into three groups.

Group I (normal arterial pCO_2). Angiograms were obtained in ten monkeys at temperatures of 36°C, 30°C, and 25°C. During the rewarming phase further angiograms were obtained at 30°C and 36°C. The arterial pCO_2 was kept constant by careful control of ventilation and frequent monitoring of arterial samples. Viscosity controlled during the cooling phase by hemodilution with saline was measured on a falling ball viscometer.

Group II (low arterial pCO_2). The method of cooling and rewarming in five monkeys was identical to that employed in Group I. Viscosity of blood was maintained constant by hemodilution during the cooling phase. The arterial pCO_2 was depressed by hyperventilation to levels of 20 mm Hg or below at 36°C, 30°C, and 25°C, and during rewarming at 30°C and 36°C. At each of these temperature levels angiograms were made at normal and low arterial pCO_2 levels.

Group III (High arterial pCO_2). Six monkeys were given an inhalation mixture of 10% CO_2 with 90% O_2 until their arterial pCO_2 reached levels of 60 mm Hg or higher. This was carried out at 36°C, 30°C, and 25°C, and

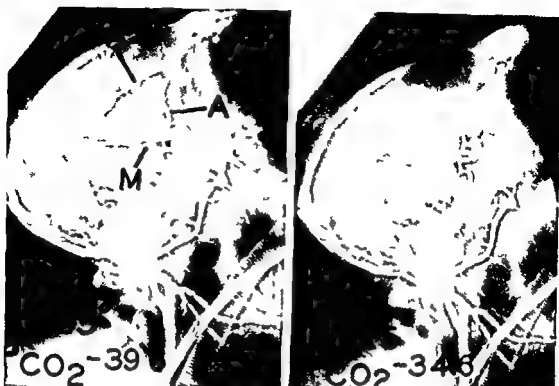


Fig. 1 Carotid arteriograms showing notable constriction of major cerebral arteries during hypothermia.

Method

Hypothermia was induced in thirty two monkeys (*Macacus cynomolgus*) by external cooling with ice to temperatures of 25° C. Eleven of these monkeys were excluded from the study because of various technical difficulties. The cooling period lasted from two to four hours depending on the weight of the monkey, and the animals were then rewarmed to normal temperatures by means of external heat. Endotracheal anesthesia was maintained by N₂O—O₂ inhalation supplemented by intravenous Pentothal[®]. A Bird respirator was used to control exchange of gases. Arterial blood pressure was monitored on a Statham strain gauge and recorded on a Grass polygraph. The electrocardiogram was continuously monitored. Samples of blood were withdrawn through a catheter placed in the right femoral artery for determinations of pCO₂, O₂ and viscosity. Core temperatures were monitored by an esophageal lead. A radiopaque polyethylene catheter with an external diameter of 0.047 inch was inserted into the left femoral artery and directed into the proximal right common carotid artery under fluoroscopic control. For each angiographic study, 2 ml of 76% Renografin[®] were injected through this catheter with

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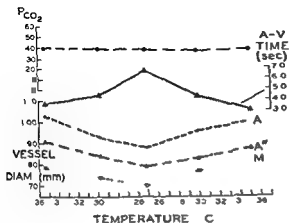


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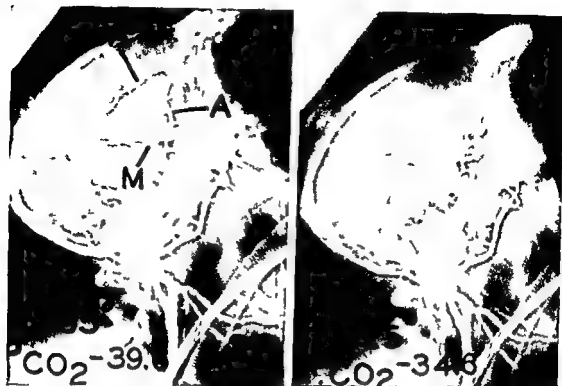


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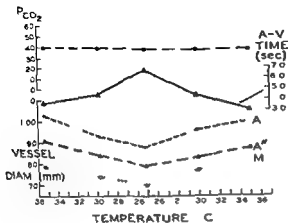


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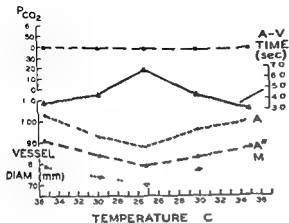


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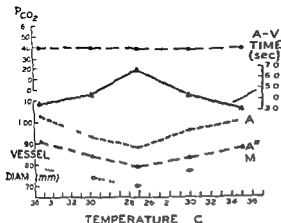


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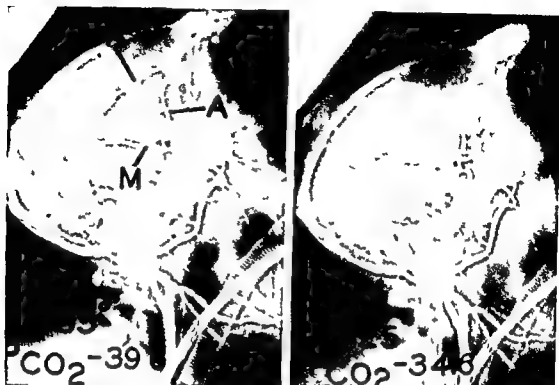


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mm to 0.88 mm at A_2 from 0.91 mm to 0.79 mm and at M from 0.79 mm to 0.70 mm. No significant or consistent hysteric effect could be observed upon rewarming.

The effect of hypothermia on circulation time was even more striking. Mean circulation time was 3.9 sec at normal temperature, 4.5 sec at 30° C and 6.7 sec at 25° C. The circulation time returned to normal upon rewarming.

Group II (low arterial pCO_2) The changes in mean size of vessels and in circulation time induced by lowering the pCO_2 levels at varying stages of hypothermia are represented in Fig. 3. Lowering of the pCO_2 resulted in a further vasoconstriction and in a prolongation of circulation time at each of the temperature levels. Mean measurements at A_1 dropped from 0.95 mm to 0.89 mm at 30° C when the mean arterial pCO_2 was lowered from 38 mm Hg to 15 mm Hg and from 0.87 mm to 0.82 mm at 25° C when the mean arterial pCO_2 was lowered from 37 mm Hg to 14 mm Hg. A concomitant prolongation in mean circulation time was observed at 30° C from 4.7 sec to 5.9 sec and at 25° C from 6.1 to 6.6 sec.

Group III (high arterial pCO_2) The effects of elevation of pCO_2 levels on mean circulation time and vessel size at varying stages of hypothermia are represented in Fig. 4. Elevation of the pCO_2 at all temperature levels resulted in increased vessel size and a more rapid circulation time. At 30° C the mean diameter of the pericallosal artery at A_1 was increased from 0.85 mm to 0.95 mm when the pCO_2 was increased from 37 mm Hg to 67 mm Hg. At 25° C an increase of the arterial pCO_2 from 38 mm Hg to 56 mm Hg resulted in a mean change at A_1 from 0.79 mm to 0.88 mm. Mean circulation time decreased from 5.0 sec to 3.1 sec at 30° C whereas at 25° C it changed from 5.9 sec to 4.1 sec.

In two monkeys the effects of prolonged anesthesia of a small catheter in the common carotid artery of repeated exposure to Renografin², and of moderate hypotension were studied. In neither were the cerebral vessel caliber or circulation time significantly changed.

Conclusions

Angiographic measurements showed a consistent constriction of the diameter of the larger cerebral arteries in response to cold despite careful control of viscosity and arterial pCO_2 . The slight concomitant drop of systemic blood pressure was shown to have no effect on the caliber of cerebral vessels or the circulation time. The percentage decrease of cerebral vessel diameter was 6 to 10% at 30° C and 11 to 15% at 25° C compared to the normothermic state. Upon rewarming the vessel caliber and circulation time returned to their previous levels.

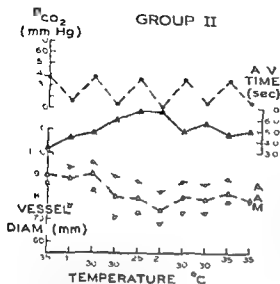


Fig. 3 Effect of lowering p_{CO_2} levels on circulation time and vessel size during various stages of cooling and rewarming

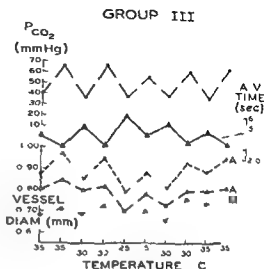


Fig. 4 Effect of elevation of the p_{CO_2} levels on circulation time and vessel size during various stages of cooling and rewarming

also, on rewarming, at 30° C and 36° C. Angiograms were obtained at each temperature level at normal and increased arterial p_{CO_2} levels. Hemodilution was used during the cooling phase to maintain viscosity of the blood at a constant level.

Two additional monkeys were maintained at normal temperature under anesthesia while a series of angiograms comparable to those of the experimental groups were obtained. Arterial blood was then withdrawn until systemic blood pressure dropped 30 mm Hg. This drop corresponded to the mean pressure drop seen at 25° C in the experimental monkeys in Groups I, II, and III. Angiograms were obtained during the hypotensive period. The blood was then returned to the monkeys and, upon return to normal blood pressure, an angiographic study was again performed.

Results

Group I (normal arterial p_{CO_2}) The changes induced in vessel diameter by hypothermia and by subsequent rewarming while normal p_{CO_2} and blood viscosity were being maintained are shown in Fig. 2. The caliber of the cerebral vessels was significantly decreased with hypothermia, even under these controlled conditions (Fig. 1) and returned to normal size when the animals were rewarmed.

Between 36° C and 25° C, the mean vessel size at A_1 decreased from 1.03

mm to 0.88 mm, at A_2 from 0.91 mm to 0.79 mm and at M from 0.79 mm to 0.70 mm. No significant or consistent hysteric effect could be observed upon rewarming.

The effect of hypothermia on circulation time was even more striking. Mean circulation time was 3.9 sec at normal temperature, 4.5 sec at 30° C and 6.7 sec at 25° C. The circulation time returned to normal upon rewarming.

Group II (low arterial pCO_2) The changes in mean size of vessels and in circulation time induced by lowering the pCO_2 levels at varying stages of hypothermia are represented in Fig. 3. Lowering of the pCO_2 resulted in a further vasoconstriction and in a prolongation of circulation time at each of the temperature levels. Mean measurements at A_1 dropped from 0.95 mm to 0.89 mm at 30° C when the mean arterial pCO_2 was lowered from 38 mm Hg to 15 mm Hg and from 0.87 mm to 0.82 mm at 25° C when the mean arterial pCO_2 was lowered from 37 mm Hg to 14 mm Hg. A concomitant prolongation in mean circulation time was observed at 30° C from 4.7 sec to 5.9 sec and at 25° C from 6.1 to 6.6 sec.

Group III (high arterial pCO_2) The effects of elevation of pCO_2 levels on mean circulation time and vessel size at varying stages of hypothermia are represented in Fig. 4. Elevation of the pCO_2 at all temperature levels resulted in increased vessel size and a more rapid circulation time. At 30° C the mean diameter of the pericallosal artery at A_1 was increased from 0.85 mm to 0.95 mm when the pCO_2 was increased from 37 mm Hg to 67 mm Hg. At 25° C an increase of the arterial pCO_2 from 38 mm Hg to 56 mm Hg resulted in a mean change at A_1 from 0.79 mm to 0.88 mm. Mean circulation time decreased from 5.0 sec to 3.1 sec at 30° C whereas at 25° C it changed from 5.9 sec to 4.1 sec.

In two monkeys the effects of prolonged anesthesia of a small catheter in the common carotid artery, of repeated exposure to Renografin[®], and of moderate hypotension were studied. In neither were the cerebral vessel caliber or circulation time significantly changed.

Conclusions

Angiographic measurements showed a consistent constriction of the diameter of the larger cerebral arteries in response to cold despite careful control of viscosity and arterial pCO_2 . The slight concomitant drop of systemic blood pressure was shown to have no effect on the caliber of cerebral vessels or the circulation time. The percentage decrease of cerebral vessel diameter was 6 to 10% at 30° C and 11 to 15% at 25° C compared to the normothermic state. Upon rewarming the vessel caliber and circulation time returned to their previous levels.

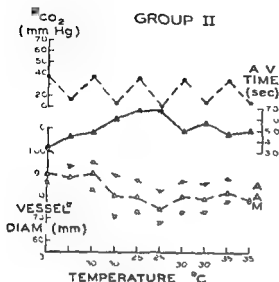


Fig 3 Effect of lowering $p\text{CO}_2$ levels on circulation time and vessel size during various stages of cooling and rewarming

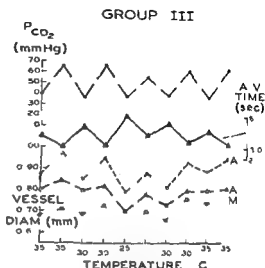


Fig 4 Effect of elevation of the $p\text{CO}_2$ levels on circulation time and vessel size during various stages of cooling and rewarming

also, on rewarming, at 30 $^{\circ}\text{C}$ and 36 $^{\circ}\text{C}$ Angiograms were obtained at each temperature level at normal and increased arterial $p\text{CO}$ levels. Hemodilution was used during the cooling phase to maintain viscosity of the blood at a constant level.

Two additional monkeys were maintained at normal temperature under anesthesia while a series of angiograms comparable to those of the experimental groups were obtained. Arterial blood was then withdrawn until systemic blood pressure dropped 30 mm Hg. This drop corresponded to the mean pressure drop seen at 25 $^{\circ}\text{C}$ in the experimental monkeys in Groups I, II, and III. Angiograms were obtained during the hypotensive period. The blood was then returned to the monkeys and, upon return to normal blood pressure, an angiographic study was again performed.

Results

Group I (normal arterial $p\text{CO}$) The changes induced in vessel diameter by hypothermia and by subsequent rewarming while normal $p\text{CO}$ and blood viscosity were being maintained are shown in Fig 2. The caliber of the cerebral vessels was significantly decreased with hypothermia, even under these controlled conditions (Fig 1) and returned to normal size when the animals were rewarmed.

Between 36 $^{\circ}\text{C}$ and 25 $^{\circ}\text{C}$, the mean vessel size at A_1 decreased from 1.03

RÉSUMÉ

L'effet de l'hypothermie sur le calibre des vaisseaux cérébraux et sur le temps de circulation a été étudié par angiographie cérébrale sur 21 singes. La $p\text{CO}_2$, la viscosité sanguine et la tension artérielle étaient mesurées. À mesure que la température baissait le calibre des vaisseaux cérébraux diminuait et le temps de circulation s'allongeait. Les modifications de la $p\text{CO}_2$ à basse température, la viscosité et la pression sanguine restant constantes, donnaient lieu aux mêmes effets physiologiques qu'à température normale.

REFERENCES

1. BERING JR. E. A., TAREN J. A., McMURREY J. D. and BERNHARD W. F. Studies on hypothermia in monkeys. II. The effect of hypothermia on the general physiology and cerebral metabolism of monkeys in the hypothermic state. *Surg. Gynec. Obstet.* 102 (1956) 134.
2. DILENCE D., DAVID M. and DILEONÉ P. Angiographic study of cerebral circulation rate during hypothermia. *Presse Méd.* 7 (1963) 1769.
3. GURDJIAN E. S., THOMAS L. M. and WEBSTER J. C. Hypothermia and pial circulation. *J. Neurosurg.* 17 (1960) 610.
4. MEYER J. S. and HUNTER J. Effects of hypothermia on local blood flow and metabolism during cerebral ischemia and hypoxia. *J. Neurosurg.* 14 (1957) 210.

Circulation time was prolonged by hypothermia despite maintenance of constant viscosity. Increased arterial pCO_2 levels could overcome this effect. The marked slowing of cerebral circulation suggests an increased resistance in the cerebrovascular bed. This is probably the result of constriction at the arteriolar level, which could not be measured angiographically.

Despite constriction induced by the hypothermic state, an increase in vessel caliber was observed with an increase in the level of arterial pCO_2 . Constriction of cerebral vessels induced by cold became more marked when the level of arterial pCO_2 was decreased below normal values.

These studies suggest that the common clinical use of hypothermia accompanied by hyperventilation, without hemodilution, may lead to decreased cerebral circulation. This slowing of cerebral circulation is of particular importance in situations frequently encountered clinically in which circulation to vital organs may already be impaired. Hyperventilation in the hypothermic state may provide ideal operative conditions. A more rapid circulation time induced by normal or elevated pCO_2 accompanied by hemodilution, however, may provide better protection of vital organs. Perhaps shrinkage of the brain frequently obtained by hyperventilation in the hypothermic state could more safely be obtained by the use of mannitol.

Acknowledgement

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SUMMARY

The effect of hypothermia on the size of cerebral vessels and on circulation time was determined in 21 monkeys by means of cerebral angiography. The arterial pCO_2 , blood viscosity, and blood pressure were controlled. The caliber of the cerebral vessels decreased and circulation time was prolonged as the temperature dropped. Alteration of the pCO_2 at low temperatures while viscosity and blood pressure were maintained constant resulted in the same physiologic effects as those seen at normal temperatures.

ZUSAMMENFASSUNG

Mittels cerebraler Angiographie wurde die Wirkung von Hypothermie auf die Weite der cerebralen Gefäße und Zirkulationszeit bestimmt. Es wurden das arterielle pCO_2 , die Blutviskosität und der Blutdruck kontrolliert. Bei Herabsetzung der Temperatur verringerte sich der Durchmesser der cerebralen Gefäße während die Zirkulationszeit sich verlängerte. Veränderungen des pCO_2 bei herabgesetzter Temperatur ergab bei konstanter Viskosität und Blutdruck dieselben physiologischen Wirkungen wie bei normaler Temperatur.



Fig 1 A small meningeal branch of the right ophthalmic artery supplies a left frontal parasagittal meningioma. Subtraction picture.



Fig 2 Right carotid angiography in a patient with a false meningioma on the left side. The tumor is supplied by a tentorial branch of the right ophthalmic artery, which is running in the free margin of the tentorium cerebelli, the midline to give off tumor vessels at its tumor attachment to the posterior bular portion of the falx.

be kept in mind that the basal dura to a large extent is supplied from the internal carotid artery as is also the dura over the frontal convexity as well as the anterior and the posterior basilar portions of the falx. These structures in or close to the midline may normally be supplied by branches from either the ipsilateral or contra lateral carotid artery and consequently tumors in these areas may also receive their vascular supply from either or both sides. Examples of tumor supply from a contra lateral carotid artery are shown in Figs 1 and 2.

In our experience a meningeal vascular supply is however not a pathognomonic sign that the lesion is a meningioma since other tumors — even malignant gliomas — may also receive part of their vascular supply from the meninges. A glioma reaching the surface of the brain often infiltrates the overlying dura and will then get part of its supply from dural vessels. This seems to be most common in temporal and basal gliomas. It is a known fact among

SIGNIFICANCE OF SOME ANGIOGRAPHIC SIGNS OF INTRACRANIAL MENINGIOMAS

by

STURE STATTIN

Cerebral angiography remains in most cases the best method of differentiating meningiomas from other intracranial expanding lesions and several more or less characteristic angiographic criteria have been advanced by various authors. The most important seem to be typical vascular displacement, tumor vessels of a characteristic appearance, vascular supply from meningeal vessels, and slow circulation through the tumor as manifested by the late filling of the drainage veins. We have earlier reported the results from a systematic investigation of the frequency of these criteria (5). In the present paper two of these criteria will be briefly discussed, namely, meningeal vascular supply and tumor circulation time. An attempt will be made to show that to some extent these criteria may be further refined but also that they are not always as pathognomonic as has often been maintained.

When looking for a meningeal vascular supply it is of importance to know that even with apparently avascular tumors it is often possible angiographically to demonstrate small vessels at the dural attachment. Most of them generally arise from the external carotid artery and for that reason separate contrast injection into the external and internal arteries is essential. It should however



Fig 1 A small meningeal branch of the right ophthalmic artery supplies a left frontal parasagittal meningioma. Same patient on picture



Fig 2 Right carotid angiography in a patient with a falx meningioma on the left side. The tumor is supplied by a tentorial branch of the right siphon which, running in the free margin of the tentorium, crosses the midline to give off tumor vessels at a tumor attachment to the posterior basilar portion of the falx.

be kept in mind that the basal dura to a large extent is supplied from the internal carotid artery, as is also the dura over the frontal convexity as well as the anterior and the posterior basilar portions of the falx. These structures in or close to the midline may normally be supplied by branches from either the ipsilateral or contralateral carotid artery, and consequently tumors in these areas may also receive their vascular supply from either or both sides. Examples of tumor supply from a contralateral carotid artery are shown in Figs 1 and 2.

In our experience a meningeal vascular supply is however not a pathognomonic sign that the lesion is a meningioma since other tumors — even malignant gliomas — may also receive part of their vascular supply from the meninges. A glioma reaching the surface of the brain often infiltrates the overlying dura and will then get part of its supply from dural vessels. This seems to be most common in temporal and basal gliomas. It is a known fact among



Fig 3 A malignant glioma in the parietal temporal region has infiltrated the dura and receives part of its blood supply from the external carotid artery. a) Internal carotid angiography b) External carotid angiography. Arrows point at the draining veins.

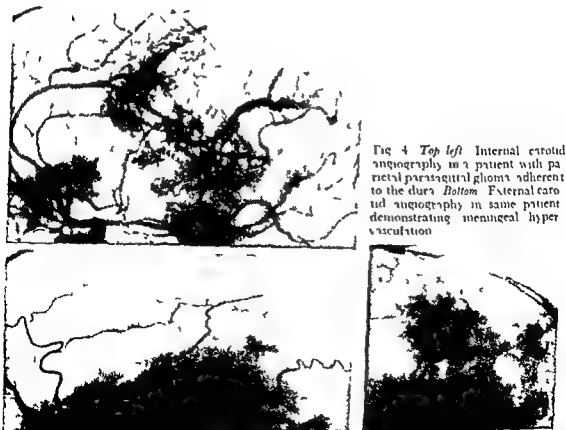


Fig 4 Top left Internal carotid angiography in a patient with parietal parasagittal glioma adherent to the dura. Bottom External carotid angiography in same patient demonstrating meningeal hyper-vascularization.

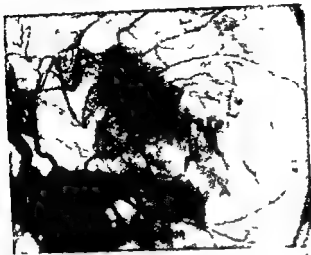


Fig 5 Falx tentorial meningioma with filling of veins in early arterial phase

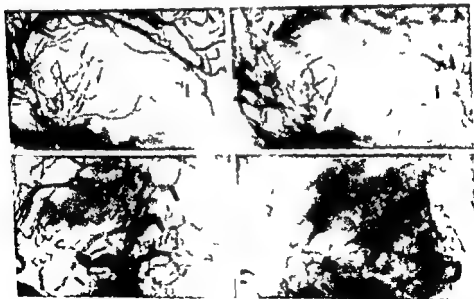


Fig 6 Top: Very early filling of veins in a patient with a basal meningioma. Lower: Same patient. Recurrence after operation. At histological examination there were still no signs of malignancy.

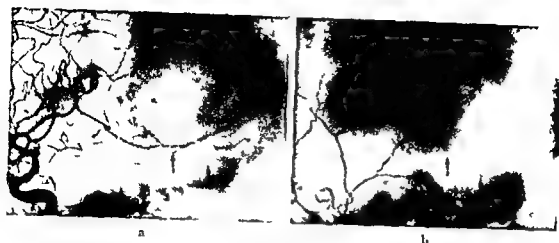


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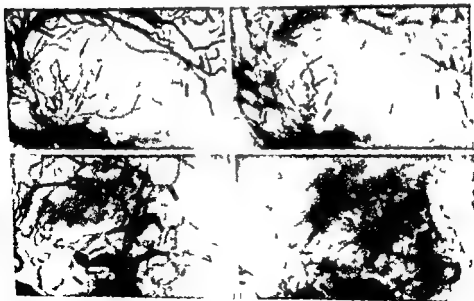


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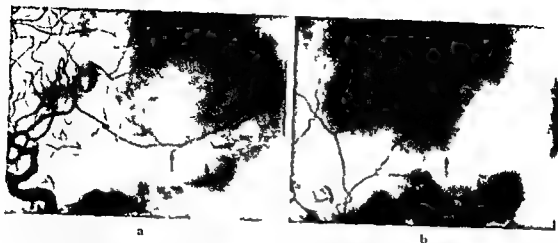


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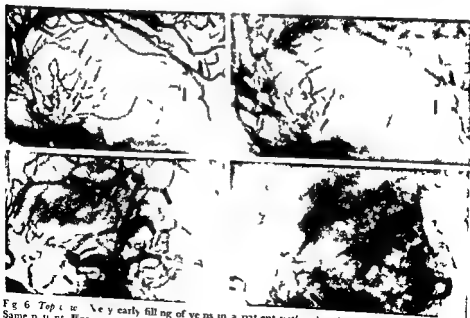


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Fig 7 Electronic subtraction pictures from an external carotid angiography series on a convexity meningioma. Small arteries are still filled and the tumor is stained with contrast medium. Arrows point at contrast already visible in a wide venous groove.

pathologists that these dura infiltrating gliomas on invasion frequently change their histological character to become more meningiomatous. Examples of gliomas supplied by meningeal vessels are shown in Figs 3 and 4.

According to most authors it is typical for meningiomas to have a long tumor circulation time with late filling of tumor veins. This criterium, although often reliable, does not always in our experience hold true. In approximately 170 intracranial meningiomas examined by angiography the tumor circulation time was found to be rather short in a number of cases. In 10 cases, veins within or on the surface of the tumor or veins draining the tumor, started to fill at approximately the same time as other intracranial veins. In 16 cases, tumor veins started to fill towards the end of the arterial filling phase before other veins were visible, and in 8 cases tumor veins filled at the beginning of the arterial filling phase, indicating arteriovenous shunts. Such very early filling of the veins may present diagnostic difficulty, especially if tumor vessels are atypical and characteristic vascular displacement is not evident. Examples of early venous filling are shown in Figs 5 and 6.

Wide venous grooves and diploic channels of the skull are a common finding in certain intracranial meningiomas. At angiography performed with ordinary technique it is hardly possible to observe contrast filling in these venous pathways. In studies with subtraction technique it has been possible in some

instances to demonstrate fairly early appearance of contrast in venous grooves (Fig. 1) and it is possible that the appearance of slow circulation through meningiomas may, at least in some cases be more deceptive than real. It is probable that introduction of more dense contrast media and further refinement of the technique including the use of subtraction methods will make it possible also to demonstrate contrast filling in venous grooves of the skull and other draining pathways in a greater number of cases.

SUMMARY

It has been found in angiographic studies that the meningeal vascular supply and the tumor circulation time are not always reliable criteria for the differentiation of meningiomas from other intracranial expanding lesions.

ZUSAMMENFASSUNG

Mitels angiographischen Studien wurde festgestellt, dass die Meningealgefäßzufuhr und die Zirkulationszeit im Tumor nicht immer zuverlässige Kriterien für die Differenzierung der Meningiome von anderen intrakraniellen Tumoren sind.

RÉSUMÉ

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REFERENCES

1. GRITZ T. A radiologic study of the brain circulation by rapid serial angiography of the carotid artery. *Acta radiol. Suppl.* 140 (1956).
2. MILETTI M. Die differentialdiagnose der Gehirngeschwülste durch die Arteriographie. *Acta neurochir. Suppl.* 1 (1950).
3. SCHIEFER W., TOIVAS W. und LOVÁRHELYI G. Die Art des Meningeoms im Gefäßbild. *Dtsch. Z. Nervenheilk.* 177 (1955) 436.
4. WICKBOM I. Angiographic determination of tumour pathology. *Acta radiol.* 40 (1953) 579.
5. WICKBOM I. and STATTIN S. Roentgen examination of intracranial meningiomas. *Acta radiol.* 50 (1958) 175.



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Mittels angiographischen Studien wurde festgestellt dass die Meningealgefäßzufuhr und die Zirkulationszeit im Tumor nicht immer zuverlässige Kriterien für die Differenzierung der Meningiomen von anderen intrakraniellen Tumoren sind

RÉSUMÉ

Des études angiographiques ont montré que l'existence d'un apport vasculaire meninge et le temps de circulation dans la tumeur ne sont pas toujours des critères fidèles pour distinguer les méningiomes d'autres tumeurs intracrâniennes

REFERENCES

- GREITZ T A radiologic study of the brain circulation by rapid serial angiography of the carotid artery Acta radiol Suppl 140 1956
- MILETTI M Die differentialdiagnose der Gehirngeschwulste durch die Arteriographie Acta neurochir Suppl 1 1950
- SCHIEFER W TONNIS W und UDVARHELYI G Die Art des Meningeoms im Gefäßbild Dtsch Z Nervenheilk 172 (1955) 436
- WICKBOM I Angiographic determination of tumour pathology Acta radiol 40 (1953) 529
- WICKBOM I and STATTIN S Roentgen examination of intracranial meningiomas Acta radiol 50 (1958) 175



Fig 7 Electronic subtraction pictures from an external carotid angiography series on a convexity meningioma. Small arteries are still filled and the tumor is stained with contrast medium. Arrows point at contrast already visible in a wide venous groove.

pathologists that these dura infiltrating gliomas on invasion frequently change their histological character to become more meningiomatous. Examples of glioma supplied by meningeal vessels are shown in Figs 3 and 4.

According to most authors it is typical for meningiomas to have a long tumor circulation time with late filling of tumor veins. This criterion, although often reliable, does not always in our experience hold true. In approximately 170 intracranial meningiomas examined by angiography the tumor circulation time was found to be rather short in a number of cases. In 10 cases, veins within or on the surface of the tumor or veins draining the tumor, started to fill at approximately the same time as other intracranial veins. In 16 cases, tumor veins started to fill towards the end of the arterial filling phase before other veins were visible, and in 8 cases tumor veins filled at the beginning of the arterial filling phase, indicating arteriovenous shunts. Such very early filling of the veins may present diagnostic difficulty, especially if tumor vessels are atypical and characteristic vascular displacement is not evident. Examples of early venous filling are shown in Figs 5 and 6.

Wide venous grooves and diploic channels of the skull are a common finding in certain intracranial meningiomas. At angiography performed with ordinary technique it is hardly possible to observe contrast filling in these venous pathways. In studies with subtraction technique it has been possible in some

instances to demonstrate fairly early appearance of contrast in venous grooves (Fig 7) and it is possible that the appearance of slow circulation through meningiomas may at least in some cases, be more deceptive than real. It is probable that introduction of more dense contrast media and further refinement of the technique including the use of subtraction methods will make it possible also to demonstrate contrast filling in venous grooves of the skull and other draining path ways in a greater number of cases.

SUMMARY

It has been found in angiographic studies that the meningeal vascular supply and the tumor circulation time are not always reliable criteria for the differentiation of meningiomas from other intracranial expanding lesions.

ZUSAMMENFASSUNG

Mittels angiographischen Studien wurde festgestellt, dass die Meningealgefäßzufuhr und die Zirkulationszeit im Tumor nicht immer zuverlässige Kriterien für die Differenzierung der Meningiomen von anderen intrakraniellen Tumoren sind.

RÉSUMÉ

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REFERENCES

1. CREITZ T. A radiologic study of the brain circulation by rapid serial angiography of the carotid artery. *Acta radiol. Suppl.* 140 (1956).
2. MILETTI M. Die differentialdiagnose der Gehirngeschwülste durch die Arteriographie. *Acta neurochir. Suppl.* 1 (1950).
3. SCHIEFER W., TONNIS W. und UDVARHELYI G. Die Art des Meningeoms im Gefäßbild. *Dtsch. Z. Nervenhk.* 172 (1955): 436.
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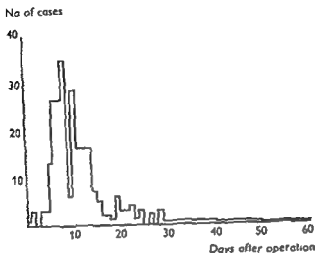


Fig 1 Block diagram showing interval between operation and angiography in 250 cases

ing to their site. Aneurysms at (or near) the origin of the posterior communicating artery (86 cases) were placed in group 1. Group 2 (61 cases) consists of aneurysms at (or near) the anterior communicating artery and group 3 (56 cases) of aneurysms on the middle cerebral artery. The cases in group 4 (29 cases) include 16 cases of aneurysm at the internal carotid bifurcation (8 left, 8 right), 10 cases of aneurysm arising from one of the pericallosal arteries and one case each of aneurysm of the basilar artery, left vertebral artery and right carotid artery near the origin of the ophthalmic artery. Multiple aneurysms comprising group 5 (18 cases) are cases in which more than one aneurysm was treated; otherwise cases of multiple aneurysm are grouped as single aneurysm according to the site of the treated aneurysm. One of the cases in group 5 was a female patient aged 49 who had a saccular aneurysm of the right posterior inferior cerebellar artery and a small arteriovenous malformation on the upper surface of the right cerebellar hemisphere; both lesions were successfully dealt with at one operation and this case has been reported elsewhere (FINE *et al.* 1960). Apart from this case and one other of middle cerebral aneurysm with supratentorial vascular malformation also dealt with at one operation, all the cases in group 5 are of multiple saccular aneurysms. In one of these cases three aneurysms were dealt with, although one was incompletely occluded. In the others two aneurysms were treated and in two of these cases one of the aneurysms was incompletely occluded.

POSTOPERATIVE ANGIOGRAPHY IN TREATMENT OF INTRACRANIAL ANEURYSMS

by

J LESLIE STEVENS

Surgical treatment of intracranial aneurysms is in many cases directed towards obliteration of the sac, but as ALLCOCK & DRAKE (1963) have pointed out, there is a scarcity of information regarding the efficacy of procedures designed to do this. As these authors observe, it is simple enough to check on the immediate results by postoperative angiography, and they reported on the findings in 70 cases in which this had been done.

The present paper deals with a further 250 cases subjected to angiography after operative treatment over a five year period from 1959 to 1963. Not in every case operated on was a postoperative angiography performed, some succumbed soon after operation before angiography could be carried out, and a few have been omitted because of incomplete documentation.

The material consists of 165 female patients and 85 male patients ranging in age from 15 to 68 years. The average age of male patients was 40 and of female patients 45. Although female patients outnumbered males by 2 to 1 this ratio was not constant in all groups, thus of aneurysms of the anterior communicating artery operated upon 33 were females, 29 males, and in the group of aneurysms of the posterior communicating artery females outnumbered males by 3 to 1.

The aneurysms in the 250 cases were distributed in five main groups accord-

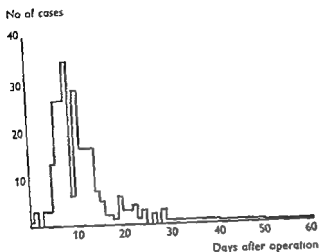


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The aneurysms in the 250 cases were distributed in five main groups accord-

Table 1
Incidence of incomplete occlusion of aneurysms

Site	Totals	Incompletely occluded	
Posterior communicating	86	13	15
Anterior communicating	61	17	28
Middle cerebral	56	7	12.3
Bifurcation of internal carotid	16	3	18.7
Pericallosal	10	0	—
Others	3	1	—
Multiple	18	3	—

although 41 (67 %) of the cases of aneurysm of the anterior communicating artery were subjected to bilateral carotid angiography postoperatively. The interval between operation and the first postoperative angiography varied from a few hours to 2 months but in 191 cases (75 %) the examination was performed within 14 days of operation. The average interval was 10 days (Fig. 1).

Surgical treatment In most cases haematoma or clot if present, was aspirated and the neck of the aneurysm was clipped with small Olivecrona clips occasionally large Olivecrona or Mayfield clips were used. In 30 cases clipping was supplemented by silk ligature packing with muscle graft or wrapping with gauze. Six aneurysms were ligated without clips in one case after clipping had failed in one of these cases however the ligature slipped off and the neck of the aneurysm was eventually clipped at a later operation. In a very few cases of aneurysm of the anterior communicating artery trapping between clips was performed or the sac was obliterated with several clips. In the process of occluding anterior communicating aneurysms and to a lesser extent middle cerebral and other aneurysms major arteries or branches were sometimes deliberately or inadvertently occluded (see below). In 5 cases of posterior communicating aneurysms and 1 case of carotid siphon aneurysm the internal carotid artery itself was permanently occluded intracranially with clips during attempts to occlude the aneurysm as the only way to control bleeding. Thirteen of the cases of multiple aneurysm were treated at one operation.

Incidence of incomplete occlusion of aneurysms An aneurysm was classified as incompletely occluded if the fundus or any part of the fundus was outlined at the postoperative angiography. CRAWFORD (1959) has shown that probably

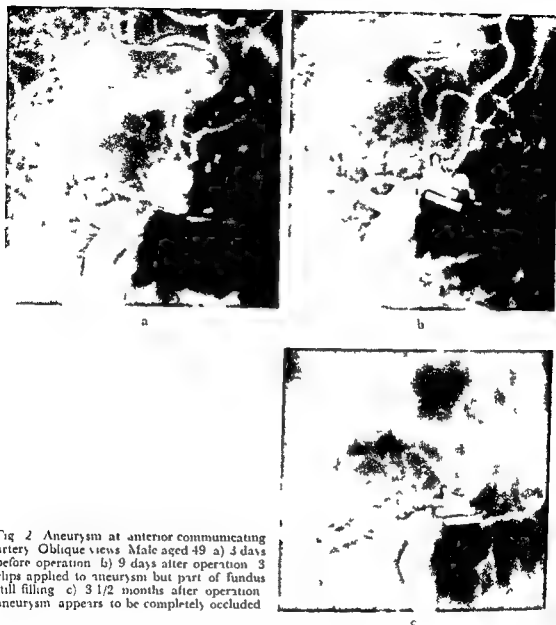


Fig. 2 Aneurysm at anterior communicating artery. Oblique views. Male aged 49. a) 3 days before operation. b) 9 days after operation. 3 clips applied to aneurysm but part of fundus still filling. c) 3 1/2 months after operation aneurysm appears to be completely occluded.

Technique and procedure

With few exceptions angiography was performed under general anaesthesia. The preoperative angiographic examination consisted of bilateral carotid angiographies (unilateral in a few cases where third nerve palsy had been the main presenting symptom) followed, usually at a later session, by unilateral or bilateral vertebral angiographies in 39 cases (15.6%). The postoperative angiography was in general unilateral and confined to the side of the aneurysm,

Table 1
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Site	Totals	Incompletely occluded	
Posterior communicating	86	13	15
Anterior communicating	61	17	28
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Bifurcation of internal carotid	16	3	18.7
Pituitary stalk	10	0	—
Others	3	1	—
Multiple	18	3	—

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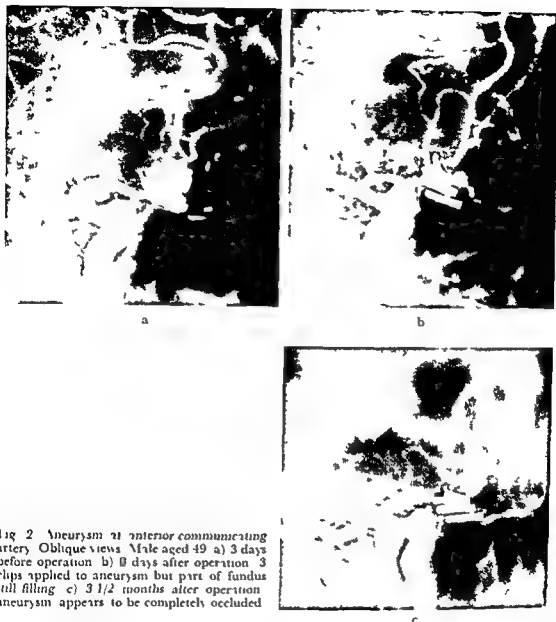


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would seem that the technical problems of occluding the sac are greatest with aneurysms in the neighbourhood of the anterior communicating artery

Late follow up angiographies (from 3 1/2 months to 4 years) were performed in 11 cases affording an opportunity to study the situation at the site of the original operation. In two cases (anterior communicating artery, Fig 2 left carotid bifurcation Fig 7) aneurysms which had been only partly occluded at the immediate postoperative angiography were shown at the later angiographies (3 1/2 and 4 months postoperatively) to be completely occluded. These cases were nevertheless classified as being incompletely occluded because it was the immediate effect of the technique of occlusion which it was desired to assess. It is possible that in these patients, re bleeding could have occurred from the remnant before occlusion became complete.

In a further case of incomplete occlusion (anterior communicating aneurysm) there was no apparent change 4 months postoperatively. In another (left middle cerebral aneurysm) some enlargement of the remaining portion of the sac was evident at 5 months postoperatively and complete occlusion was eventually secured at a second operation (Fig 3).

In the remaining 7 cases the aneurysms which had been shown to be completely occluded at the immediate postoperative angiography showed no recurrence in angiographies performed at periods of 6 months (2 cases, posterior communicating), 8 months (posterior communicating) 1 year (middle cerebral) 1 year and 3 months (anterior communicating) 1 year and 6 months (posterior communicating) and 4 years (middle cerebral). However in one of these cases which had been re examined by carotid and vertebral angiography because of recurrence of subarachnoid haemorrhage although no further aneurysm was found there appeared to be some dilatation of the carotid artery at the site of the previously clipped aneurysm (Fig 4). Re exploration showed no evidence of bleeding in this area. In one case a part of the neck was outlined at the first postoperative angiography and no change was evident at the later angiography (Fig 5).

Postoperative clinical complications. It is generally assumed that occlusion or spasm of a major artery or its branches may cause ischaemic damage to the part of the brain supplied by the affected vessel as both these appearances were found to be common after intracranial operations on aneurysms an attempt was made to correlate the angiographic appearances with the patient's postoperative clinical state at the time of the check angiography. Some difficulty was experienced in classifying clinical states but it was decided for the purposes of this study to divide the cases into those which showed no postoperative deterioration and those which did, such deterioration was assessed

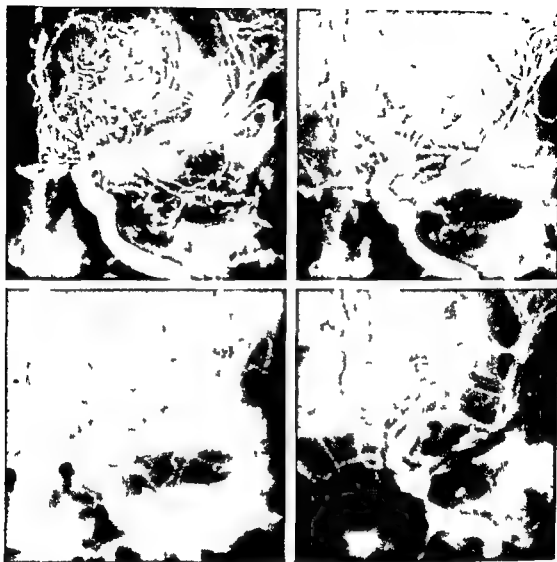


Fig. 3. Aneurysm of left middle cerebral artery. A. p. views. Female aged 49. *Top left* 2 days before operation. *Top right* 9 days after operation clip on neck aneurysm not occluded but reduced in size. *Lower left* 5 months after operation aneurysm has increased in size. *Lower right* 12 days after the angiogram at lower left and 7 days after second operation further clip applied aneurysm completely occluded.

only 2 or 3 % of aneurysms bleed from the neck and in this study therefore an aneurysm was regarded as successfully occluded even when the neck proximal to the clip was outlined postoperatively. On this basis, incomplete occlusion was found in 44 cases of the series an overall incidence of 17.6 %, which corresponds closely with the figure of 18.5 % found by ALLCOCK & DRAKE in their series. Table 1 shows the incidence of incomplete occlusion in the main sites. While an aneurysm at any site presents its own difficulties it

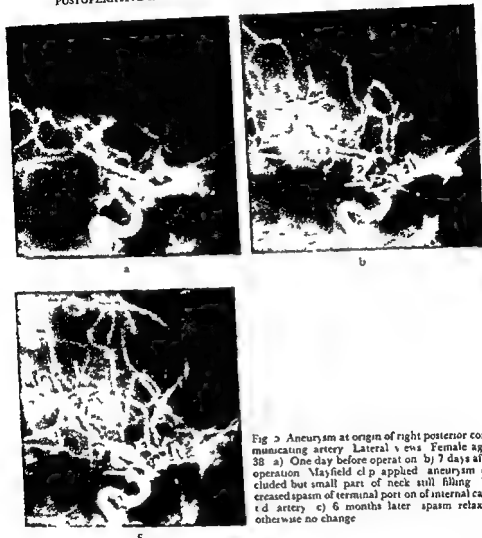


Fig 3 Aneurysm at origin of right posterior communicating artery. Lateral views. Female aged 38. a) One day before operation. b) 7 days after operation. Mayfield clip applied aneurysm occluded but small part of neck still filling. Increased spasm of terminal portion of internal carotid artery. c) 6 months later spasm relaxed otherwise no change.

one or both anterior cerebral arteries and as such studies were performed in only 67 % of cases of aneurysm of the anterior communicating artery a few cases of postoperative vascular occlusion were possibly not discovered. There was evidence of occlusion of a main artery or one or more of its major branches in 60 cases of the series an incidence of 24 %. In 40 cases the surgeon had either deliberately occluded the vessel or was aware that he might have occluded a major branch. In 23 cases one of the pericallosal arteries was occluded, and in 5 cases both these arteries were occluded. In 9 cases one middle cerebral

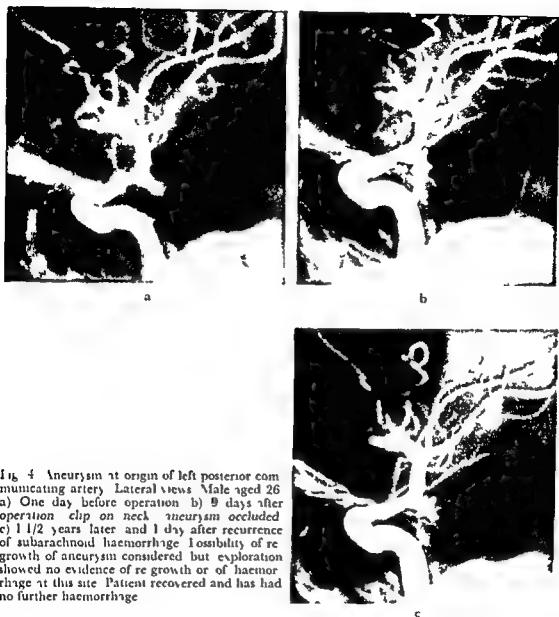


Fig. 4 Aneurysm at origin of left posterior communicating artery. Lateral views. Male aged 26 a) One day before operation b) 9 days after operation clip on neck aneurysm occluded c) 1 1/2 years later and 1 day after recurrence of subarachnoid haemorrhage. Possibility of re-growth of aneurysm considered but exploration showed no evidence of re-growth or of haemorrhage at this site. Patient recovered and has had no further haemorrhage.

ed by the development or exacerbation since operation of neurological signs, eg, hemiparesis, hemiplegia, and dysphasia, and signs of mental upset eg, confusion and akinetic mutism. In a great many considerable improvement or complete recovery occurred in the period following the postoperative angiography, but this was not taken into account.

Vascular occlusion. Without cross compression studies and, if necessary, bilateral angiography, it may not be possible to demonstrate unequivocal occlusion of



Fig. 1. Aneurysm at origin of right posterior communicating artery. Anteroposterior views. Female aged 44. a) 2 days before operation. b) 31 days after operation. 4 clips occluding internal carotid artery; considerable reduction of lumen of proximal part of artery.

formly reduced to half or less the preoperative diameter (Fig. 6) and the phenomenon is presumably associated with the considerably reduced flow.

Spasm. The series offered an opportunity to study the incidence of spasm as with two or more angiographies separated by an interval of several days even small alterations in vessel calibre could be easily detected. Of the 191 cases which had their first check angiography within 14 days after operation 114 (59.7%) showed new or increased spasm, 24 cases (12.5%) showed some degree of relaxation and in 31 cases (16.2%) there was considered to be no spasm either before or after operation.

The presence of spasm was clearly related to the interval since operation, the earlier the angiography the more commonly was spasm found and the more severe it was. Thus in every one of 10 cases in which angiographies were performed within 5 days of operation new or increased spasm was noted, severe or extensive in six. In all 16 cases considered to have severe postoperative spasm the angiographies were performed within 11 days of operation (Fig. 7). There was a reduction in the incidence of new or increased spasm from 100%

Table 2

Relationship of postoperative vascular occlusion to patient's postoperative condition (3 main groups)

Site	Totals	Cases with postoperative vascular occlusion	
		Clinical deterioration	No deterioration
Posterior communicating	86	5 (5.8%)*	1 (1.2%)
Anterior communicating	61	11 (18%)	9 (14.7%)
Middle cerebral	56	13 (23.2%)	9 (16%)

* Includes one case of occlusion of middle cerebral artery

artery, and, in 16 cases, one or more branches of this artery were occluded. In 7 cases an internal carotid artery was occluded — in 6 deliberately (as noted above) and in 1 probably as a consequence of clipping the middle cerebral artery near its origin in a case of aneurysm in the region of the carotid bifurcation, where, after a clip had been applied to its neck, the aneurysm tore off the parent vessel.

Of the 60 cases showing vascular occlusion, 34 (56.6%) had evidence of clinical deterioration and in the remaining 190 cases in which no definite vascular occlusion could be detected 71 (37.3%) had evidence of deterioration. Table 2 shows the relationship of postoperative vascular occlusion to the patient's postoperative state in the three commonest aneurysmal sites.

These findings suggest that deterioration with vascular occlusion is not related to the aneurysm which is treated but to the vessel occluded; the high incidence of deterioration in the posterior communicating group reflects the tendency for vascular occlusion, when it occurs with aneurysms at this site, to affect the internal carotid artery itself. While there is obviously a greater risk of deterioration with vascular occlusion in general, the relationship is not consistent and one may infer that other lesions may be responsible for deterioration, in cases with and without evidence of occlusion. Even the presence of collateral circulation may not prevent the occurrence of signs of vascular deficiency. For example, of the 7 cases of internal carotid occlusion, 4 showed severe neurological deficit postoperatively in spite of collateral circulation having been demonstrated angiographically, preoperatively in two, and postoperatively in the other two. In the 6 cases where the internal carotid artery had been clipped there was reduced calibre of the internal carotid artery from its origin to the level of occlusion at the siphon. The lumen of the vessel was uni-



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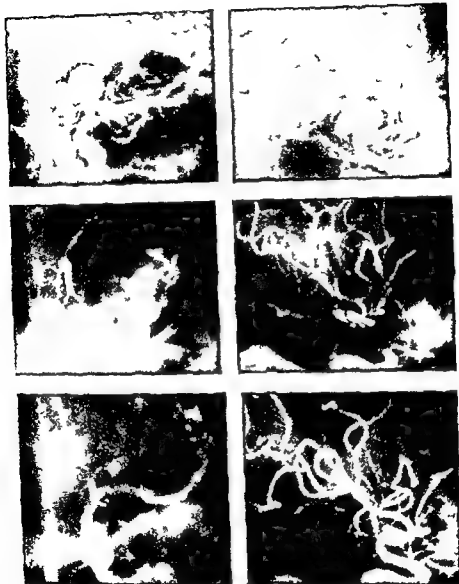


Fig 7 Large aneurysm at bifurcation of left internal carotid artery. A p films on left lateral films on right. Upper row 6 days before operation. Center row 9 days after operation aneurysm not completely occluded severe spasm. Patient well no neurological signs. Lower row 4 months after operation aneurysm completely occluded relaxation of spasm. Left internal carotid artery may be occluded.

Table 3

Relationship of postoperative spasm to patient's postoperative condition

Site	Cases with increased spasm*		No increase of spasm*	
	With deterioration	Without deterioration	With deterioration	Without deterioration
Posterior communicating	12 (11 %)	32 (37 %)	10 (11.6 %)	26 (30.7 %)
Anterior communicating	14 (22.9 %)	9 (14.7 %)	8 (13.1 %)	10 (16.4 %)
Middle cerebral	5 (8.9 %)	11 (20 %)	6 (10.7 %)	12 (21.4 %)

* Excluding cases with vascular occlusion

in the first 5 post operative days to 61 % of 105 angiographies from 6 to 10 days after operation, 50 % of 83 angiographies from 11 to 15 days, and 43 % of 30 angiographies from 16 to 25 days after operation. These findings suggest that operation in cases of subarachnoid haemorrhage provokes or aggravates spasm. DU BOULAY (1963), however, considered that there was no direct evidence that generalised spasm was provoked or prolonged by operation, but in the 24 cases in his series it appears that the average interval between operation and check angiography was 17 days as against 10 days in the present series, and this difference may explain the apparently contradictory conclusions.

Excluding cases with evidence of vascular occlusion there were 104 cases showing new or increased spasm postoperatively, in 41 of these (39.4 %) there was clinical deterioration. Similarly, of 86 cases showing no fresh spasm after operation, 31 (36.0 %) had clinical deterioration. Table 3 compares the incidence of deterioration with spasm in aneurysms at the three main sites. Only with anterior communicating aneurysms was there any correlation between increased spasm and clinical deterioration and then it was not very striking. In the 16 cases with severe spasm there were 7 which showed definite deterioration but in the remainder there was either improvement or no deterioration. On the other hand, of the 24 cases in which there was some relaxation of spasm, 11 cases (46 %) showed varying degrees of deterioration (although in 4 of these there was also evidence of vascular occlusion). Of the 31 cases considered to have no spasm before or after operation 5 cases (16 %) showed some deterioration (vascular occlusion in one case). It seems reasonable to conclude from these findings that although operation may aggravate or provoke spasm in cases of subarachnoid haemorrhage, such spasm does not appear to have such damaging effects as are generally attributed to the spasm of spontaneous preoperative haemorrhage.

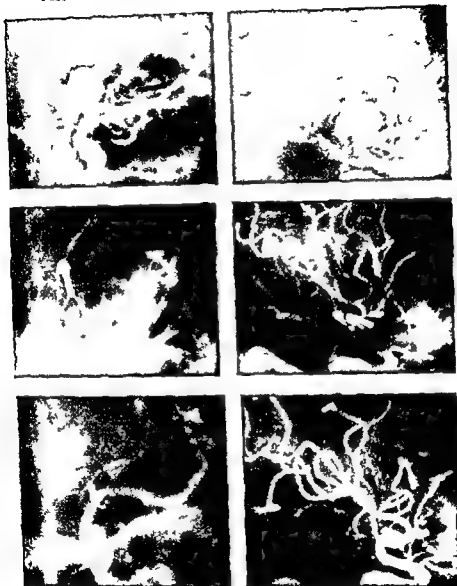


Fig 7 Large aneurysm at bifurcation of left internal carotid artery. A p films on left lateral films on right. Upper row 6 days before operation. Centre row 9 days after operation aneurysm not completely occluded severe spasm. Patient well no neurological signs. Lower row 4 months after operation aneurysm completely occluded relaxation of spasm. Left anterior cerebral artery may be occluded.

Acknowledgements

I wish to thank my surgical colleagues, J. Sloan Robertson, J. Schorstein, A. Paterson and W. B. Jennett for giving me access to the records of their patients and for advice and encouragement.

SUMMARY

A series of 250 cases of intracranial aneurysms treated by operative occlusion have been studied with postoperative angiography to elucidate the incidence of incomplete occlusion of the aneurysms as well as postoperative vascular occlusion and spasm. An attempt has been made to assess the extent to which these latter complications may affect the patient's post-operative clinical condition.

ZUSAMMENFASSUNG

Eine Serie von 250 Fällen von intrakraniellen Aneurysmen die operativ verschlossen worden waren wurden angiographisch untersucht um das Vorkommen von unkompletten Verschluss wie auch postoperativem Verschluss und Spasmus zu untersuchen.

RÉSUMÉ

Pour déterminer la fréquence de l'occlusion chirurgicale incomplète des anévrysmes de l'occlusion vasculaire post opératoire et du spasme l'auteur a examiné par angiographie post opératoire une série de 250 cas d'anévrysmes intracrâniens traités par occlusion chirurgicale. Il a essayé de déterminer dans quelle mesure ces complications peuvent influencer sur l'état clinique post opératoire du malade.

REFERENCES

- ALLOCK, J. M. and DRAKE, C. G. Post operative angiography in cases of ruptured intracranial aneurysm. *J. Neurosurg.* 20 (1963) 752.
 DU BOULAY, G. Distribution of spasm in the intracranial arteries after subarachnoid haemorrhage. *Acta radiol.* 1 (1963) 257.
 CRAWFORD, J. Symposium on intracranial aneurysms — pathological aspects. *Brit. J. Radiol.* 32 (1959) 84.
 FINE, R., PATERSON, A., STEVEN, J. L. and GAYLOR, J. B. Simultaneous occurrence of intracranial aneurysm and arterio-venous malformation. *Brit. Med. J.* 2 (1960) 711.

COMPARISON OF POSTOPERATIVE ANGIOGRAPHIC APPEARANCE AND CLINICAL SIGNS IN ARTERIAL ANEURYSM OF THE INTRACAVERNOUS INTERNAL CAROTID ARTERY

by

LAWRENCE STRENGER

According to JEFFERSON (13) the first complete clinical picture of a non fistulous intracavernous carotid aneurysm was described by BARTHOLOW (4) in 1872 In 1869 ADAMS (1) published A case of aneurysm of the internal carotid artery in the cavernous sinus causing paralysis of the third fourth fifth and sixth nerves Many (5 7 8 10 13 20) since then have written of the syndrome of the lateral wall of the cavernous sinus

Certain radiological signs have been frequently seen with intracavernous aneurysms and are considered by some to be classical (2) The earliest appreciated (5 12 19) changes were adventitious calcifications (as Albi's rings) later the significance of associated erosive phenomenon was appreciated (2 15 16 17 18) LOMBARDI and his associates (15) recently summarized the changes noted in their series

Although it may not be true in all cases (15 17) angiography generally offers the sine qua non in the establishment of the diagnosis of an intracavernous aneurysm It is frequently used to illustrate the response of the lesion to



Fig 1 Case 1 Angiograms taken before operation (a) and afterwards (b)

therapy. The following cases demonstrate the failure of correlation between the postoperative angiographic appearance of the aneurysm and the clinical signs.

Case reports

Case 1 A 54 year old Spanish speaking housewife from Puerto Rico was admitted to St Vincent's Hospital complaining of progressive diplopia of six months duration. She demonstrated a left lateral rectus paresis, a ptotic left eyelid with anisocoria (the left pupil being larger than the right). Routine studies including cerebrospinal fluid examination were unremarkable. A large saccular aneurysm of the intracavernous portion of the left internal carotid artery was demonstrated by angiography (Fig 1 a). Adequate cross filling was suggested by the cross compression studies and was confirmed by the Matas Test. Three days later a left common carotid ligation was performed. On examination the next morning, a total left ophthalmoplegia was in evidence. The pupil was dilated and fixed and the lid completely ptosed. The second day she complained of severe left sided head pain which persisted.

On the 13th postoperative day a left carotid angiography was performed (Fig 1 b). This demonstrated minimal filling at the site of the sac with the patient in the supine position. In order to obviate chances of failure of filling of the aneurysm because of the layering effect additional films were taken with the patient in the prone position. The ophthalmoplegia and left frontal pain persisted until the time of discharge 11 weeks after the operation.

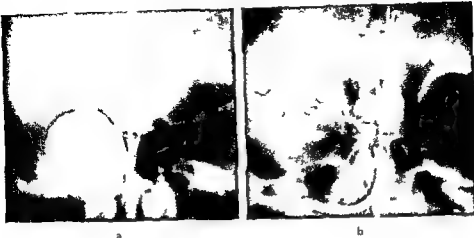


Fig 2 Case 2 Angiograms taken before operation (a) and afterwards (b)

Case 2 A 55-year old West Indian Negress was admitted to St Vincent's Hospital complaining of right retro-orbital pain of two weeks duration. This began suddenly and was of progressive severity. Further inquiry elicited that her right eye began turning in approximately three years prior to the time of her admission.

Examination revealed an obese, anxious, hypertensive negress with a blood pressure of 210/120 mm Hg. She demonstrated loss of visual acuity of the right eye (a right superior nasal quadrant defect on visual field examination), pallor of the right optic disc and a depressed right corneal reflex. There was also a right lateral rectus palsy. The routine skull films were suggestive of the presence of an intracranial aneurysm. This was confirmed by angiography (Fig 2 a). A series of films taken with cross compression suggested the adequacy of cross circulation and was supported by the Matas Test. Common carotid ligation was performed. The patient refused postoperative angiography at this admission.

Approximately three weeks later she returned to the hospital complaining of severe right retro-orbital pain. The right eye was now totally amblyopic and additional extraocular palsies were noted. Angiography at this time revealed non-filling of the aneurysmal sac (Fig 2 b). The pain abated after three months but she did not recover vision or any significant extraocular movements.

Case 3 A 44-year old American white female was investigated for headache at Jefferson Medical College in 1962. A carotid angiography was performed on the right side revealing a small intracranial aneurysm. Subsequently a right common carotid ligation was performed. There was remission of the head pain but it returned a few weeks later with increased severity. She presented herself approximately two years later complaining of syncopal episodes and right frontal head pain. Examination revealed hypesthesia over the ophthalmic division of the right trigeminal and a mild paresis of the right medial rectus muscle. She was admitted to the Shore Memorial Hospital and a right carotid angiography was performed 18 months after the first investigation. No aneurysm or other intracranial mass was identified.

Discussion

DANDY (6) and HAMBY (11) felt that orbital pain was highly suggestive of enlargement of the intracavernous aneurysms. The cases presented demonstrate that collapse or thrombosis of the sac, as demonstrated angiographically, may lead to the same complaint. The associated ocular and extraocular muscle signs may also be aggravated. Others (9, 14) have reported similar situations.

In these cases the mechanics of production of the signs is obscure. The collapsing sac may have caused traction upon the dura of the lateral wall of the cavernous sinus affecting the adjacent structures. The wall of the lateral sinus is innervated by the nervus meningus medius arising principally from the trigeminus. The implications of pain referral are obvious. However, the fact that the aneurysmal sac does not fill with contrast medium at postoperative angiography does not necessarily mean it has reduced at all in size. The entire sac may be filled with thrombus. This might suggest that ischemia of the area is being produced by involvement of the small vessels arising from the intracavernous carotid artery, as described by PARKINSON. This explanation seems less tenable than the former considering the available anastomosis, and the apparent patency of adjacent vessels. The pathophysiology is, as stated, enigmatic.

SUMMARY

Three cases of intracavernous arterial carotid aneurysms treated by common carotid ligation are presented and the failure of correlation between angiographically demonstrated resolution of the lesions and improvement in clinical signs is discussed.

ZUSAMMENFASSUNG

Drei Fälle von intrakavernösem Carotisaneurysma wurde mit Carotisligatur behandelt. Die Diskrepanz zwischen angiographischem Befund und dem Verlauf der klinischen Symptome wird besprochen.

RÉSUMÉ

L'auteur présente trois cas d'anévrisme carotidien intracaverneux traités par ligature de la carotide primitive. Il étudie la discordance entre la disparition de la lésion prouvée par l'angiographie et les signes cliniques.

REFERENCES

1. ADAMS J. Aneurysms of the internal carotid in the cavernous sinus. *Lancet* 2 (1869): 768.
2. ALAJOUANINE T, THURAL R, NEHLIL J et BALDACCY Y. L'anévrisme du siphon carotidien et son retentissement osseux (Réunion du trou optique et de fente sphénoïdale). *Rev. neurol.* 92 (1955): 249.

- 3 ALBL H Aneurysma der Carotis Interna einen Hypophysentumor vortäuschen Ein Beitrag zur Diagnose Intrakraniellen Aneurysmen Fortschr Röntgenstr 39 (1939) 890
- 4 BARTHOLOW R Aneurysms of the arteries at the base of the brain Their symptomatology diagnosis and treatment Amer J med Sci 64 (1872) 373
- 5 DANDY W E Intracranial arterial aneurysms in the carotid canal Diagnosis and treatment A M A Arch Surg 45 (1942) 335
- 6 — Intracranial arterial aneurysms Comstock Publishing Co Ithaca N Y 1944
- 7 DAVIS L E Lesions of the paratrigeminal area J Amer med Ass 80 (1923) 380
- 8 — and MARTIN J Surgical lesions of the paratrigeminal area J Amer med Ass 113 (1939) 1592
- 9 FISHER R G and GORAN A Thrombosis of an intracranial aneurysm and cervical portion of the internal carotid artery in a child J Neurosurg 18 (1961) 698
- 10 FOXC Syndrome de la paroi externe du sinus caverneux Rev neurol 38 (1922) 827
- 11 HAMBY W H Intracranial aneurysms Charles C Thomas Springfield Ill 1952
- 12 HEUER G J and DANDY W E Roentgenography in the localization of brain tumors based upon a series of one hundred consecutive cases Bull Johns Hopk Hosp 27 (1916) 311
- 13 JEFFERSON G On the saccular aneurysms of the internal carotid in the cavernous sinus Brit J Surg 26 (1938) 267
- 14 LEVITT J M Aneurysm of the internal carotid artery simulating tumor Report of a case Arch Ophthalmol 43 (1950) 500
- 15 LONBARDI G PASERINI A and MIGLIAVACCA F Intracavernous aneurysms of the internal carotid artery Amer J Roentgenol 88 (1963) 361
- 16 McINNEY J ACREE T and STOLTZ S Syndrome of the unruptured aneurysm of intracranial portion of internal carotid artery Bull Neurol Inst 5 (1936) 247
- 17 MEADOWS S P Intracavernous aneurysms of the internal carotid artery Their clinical features and natural history Arch Ophthalmol 69 (1959) 566
- 18 RUSCHBIETH R H C and BULL J W D Significance of enlargement of superior orbital (sphenoid) fissure Brit J Radiol 31 (1958) 125
- 19 SOSMAN M C and VOGT E C Aneurysms of the internal carotid artery and the Circle of Willis from a roentgenological viewpoint Amer J Roentgenol 15 (1926) 122
- 20 STOPFORD J H H The arteries of the pons and medulla oblongata III J Anat 51 (1917) 250

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Fig 1 Angiogram showing slightly angioblastic meningioma of the olfactory groove with typical meningioma arrangement of the vessels (group II) without obvious embolizations. It cannot be differentiated from a benign meningioma.



Fig 2 Angiography. A slightly angioblastic meningioma arising from the anterior clinoid process. Wheel spoke arrangement of the tumour vessels as in a benign meningioma but also slight irregularities of the same vessels (lumen variations, small pathologic connections) suggesting the presence of angioblastic growth (group II).

Serafimerlasarettet in Stockholm. All cases are histologically verified, most of them by one and the same experienced pathologist.

In the angiographies performed before 1952 only slow series were used. From 1952 to 1955 both slow and rapid series, and ever since 1955 only rapid series, which in this material means 10 films at a rate of two films per second immediately followed by 10 films at a rate of one film per second.

The material available consists of 46 cases, which I have subdivided into the following four groups on the basis of the angiographic appearance of the tumour vessels: group I, tumours angiographically poor in vessels (9 cases, approx. 20%); group II, tumour vessels with typical meningioma arrangement (10 cases, 20%); group III, tumours moderately rich in irregular vessels (16 cases, 35%); group IV, tumours very rich in irregular vessels (11 cases, 25%). In no case was the angiographic appearance normal.

In group I dislocation of vessels outside the tumour could be seen in the angiograms, while the tumour itself appeared poor in vessels. In the remaining three groups at least one feeding artery was always a branch from the external carotid artery, which means that for all practical purposes the diagnosis of a

ANGIOGRAPHIC APPEARANCE OF ANGIOBLASTIC MENINGIOMAS

by

RUNE TELENUS

In a meningioma the tumour vessels are lined either by endothelial cells or directly by tumour cells. In the latter case the histologic structure is said to be angioblastic. If at least some part of a meningioma is found to have such an angioblastic structure the meningioma is classified as angioblastic, even if most of the tumour vessels are lined by ordinary endothelium. This definition of an angioblastic meningioma is the one used in this paper (cf BERGSTRAND & OLIVECROVA 1935, CUSHING & EISENHARDT 1938, O. T. BAILEY 1940, P. BAILEY 1948, ZULCH 1956).

As is well known the angioblastic meningiomas have a bad prognosis compared to ordinary meningiomas (cf BERGSTRAND & OLIVECROVA, CUSHING & EISENHARDT), and as a rule the more widespread the angioblastic way of growth in the tumour, the more difficult is the operation and the worse is the prognosis. It is therefore of definite value to be able to diagnose a meningioma as angioblastic before the operation, and also if possible to determine the extent of the angioblastic growth.

The present investigation consists of a study of carotid and vertebral angiographies of cases of angioblastic meningiomas operated upon since 1951 at



Fig 5 Angiogram of large recurrence of a highly angioblastic convexity meningioma. The angiogram shows strikingly all types of irregularities of the tumour vessels (pathologic vessels of mal gnar cy) but without rapid passage from feeding arteries to drainage veins. The circulation time through the tumour was the same as outside the tumour. No capillary blush.

meningioma (not necessarily angioblastic) was definite in about 80 % of the whole material.

Group II (cf MONIZ 1940, LORENZ 1940, WICKBOM 1948, 1953, WICKBOM & STATTIN 1958, LIMA 1950, MILETTI 1950, LINDCREN 1954, SCHIEFER & TONNIS 1954, SCHIEFER et coll. 1955) will be illustrated by two typical cases.

Fig 1 shows an angioblastic meningioma of the olfactory groove where the feeding meningeal branches enter the center of the tumour and the cerebral feeding arteries the periphery without any irregularity of the tumour vessels. It cannot be differentiated from an ordinary non angioblastic meningioma (in fact it was only slightly angioblastic).

Fig 2 represents an angioblastic meningioma of the anterior clinoid process with the typical wheel spoke arrangement of the tumour vessels. But in this case there are slight irregularities with lumen variations of the tumour vessels and pathologic connections between them giving rise to small irregular sinuses in the tumour suggesting that this is not an entirely ordinary meningioma but an angioblastic one.

In group III the irregularities of the tumour vessels are more apparent

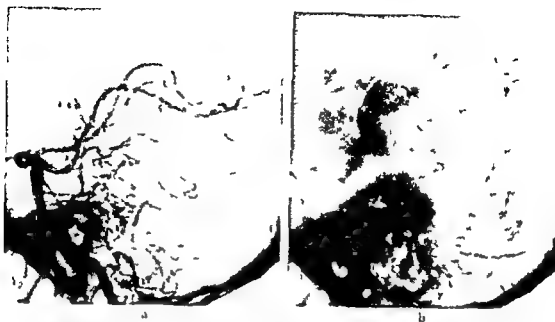


Fig 3 Angiography a) A moderately angioblastic meningioma infiltrating the tentorium (group III) The irregularity of some of the vessels is more apparent than in group II b) Irregular capillary blush



Fig 4 Angiogram showing a highly angioblastic meningioma of the posterior fossa. The tumour vessels are very tortuous and very irregular in details with lumen variations and pathologic connections (group IV) (Later on the contrast medium passed over into a longstanding capillary blush. No arteriovenous shunting through the tumour)

Table

Correlation between histologic and angiographic findings

Histologic group (I—IV)	Angiographic group (I—IV)			
	I	II	III	IV
1 Extensive regressive changes	6	1	2	= 9
2 Slightly angioblastic	3	6	2	= 11
3 Moderately angioblastic		2	11	4 = 17
4 Highly angioblastic		1	1	7 = 9
	9	10	16	11 = 46

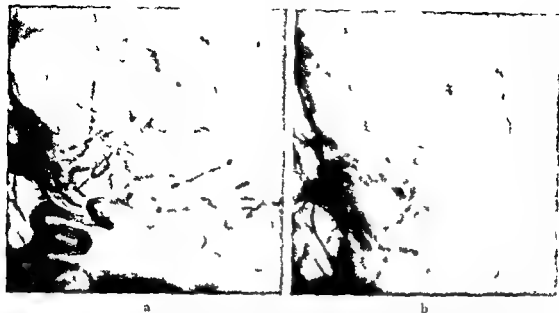
30 cases i.e. about 65 % of the whole material had a longstanding capillary blush with prolonged circulation time. The absence of rapid arterio-venous shunts is of course valuable for the differential diagnosis between angioblastic meningiomas and other malignant intracranial lesions. (For a more detailed discussion see HENNINGSSON 1939, ENGESET 1944, WICKBOM 1948, 1953, MILETTI 1950, SCHIEFER, TONNIS & UDVARHELYI 1954, 1955, GREITZ 1956, BERNASCONI & CASSINARI 1958, ZACHARISOV 1963, STATTIN 1964.)

Correlation between histologic and angiographic findings. By separating those tumours in which the regressive histologic changes were extensive (histologic group I in the Table) from the rest of the cases and then grading the latter according to the extent of the angioblastic mode of growth into slightly, moderately and highly angioblastic meningiomas (histologic groups 2—4 in the Table) it has been possible to correlate four histologic groups with the four above mentioned angiographic groups I—IV. The result is shown in the Table. As is well seen from this table the correlation between corresponding histologic and angiographic groups 1—I, 2—II, 3—III and 4—IV is surprisingly good.

Conclusions

1 A positive correlation exists between the histologically found extension of the angioblastic structure in the tumour and the angiographically estimated degree of irregularity of the tumour vessels (irregularity in the sense described above: irregular course and arrangement of tumour vessels compared to ordinary meningiomas: lumen variations, pathologic connections between vessels within the tumour, often with formation of small sinuses).

2 If the angioblastic meningioma is poor in vessels extensive regressive



a

b

Fig 6 Angiograms from the only case in the material where the circulation time was shortened. In lower part of tumour there are small irregular vessels. In upper part a pathologic connection between two arteries giving rise to an irregular sinus from which the contrast medium successively but not very rapidly passed over into a system of capsular veins (b) and from them to a drainage vein which was filled somewhat earlier than other intracranial veins. No capillary blush.

Fig 3 shows two films from one and the same angioblastic meningioma infiltrating the tentorium, Fig 3 a shows the moderately irregular feeding arteries and tumour vessels, and Fig 3 b the following capillary blush, more irregular than what is usually the case in an ordinary meningioma.

In group IV the irregularities of the vessels are striking. Fig 4 shows the very tortuous tumour vessels of a highly angioblastic meningioma in the posterior fossa. Fig 5 is a recurrence of a huge angioblastic convexity meningioma with widespread pathologic connections between the irregular tumour vessels.

Fig 6 shows films from the only case (also belonging to group IV) in the entire material where a comparatively rapid passage of contrast medium from arteries to veins could be found. Fig 6 a shows a pathologic connection between two arteries within the tumour, and Fig 6 b capsular veins filled earlier than other intracranial veins. However, no very rapid passage of the contrast medium directly from a preformed feeding artery into a preformed drainage vein — a useful definition of the so called 'rapid arterio venous shunt' — was found in this case or in any other case in the material. On the contrary — in spite of the more or less malignant nature of the angioblastic meningiomas — all the other cases in this material had a circulation time (for definition see GREITZ 1956) equal to or longer than the rest of the intracranial vessels. As many as

REFERENCES

- BAILEY O T Histology of meningiomas Arch Path 30 (1940) 42
- BAILEY P Intracranial tumours Ch C Thomas Springfield 1948
- BERSTRAND H and OLIVECROVA H Angioblastic meningiomas Amer J Cancer 24 (1935) 377
- BERASCONI V e CASSINARI V Studio angiografico di 207 casi di meningiomi sopratentoriali operati G Psychiat Neuropat 86 (1958) 63
- CUSHING and EISENHARDT Meningiomas their classification regional behavior life history and surgical end results Ch C Thomas Springfield and Baltimore 1938
- ENGESST A Cerebral angiography with perabrodil Acta radiol Suppl 56 (1944)
- GREITZ T A radiologic study of the brain circulation by rapid serial angiography of the carotid artery Acta radiol Suppl 140 (1956) 76 84
- HENNINGSSON H Arteriographic diagnosis of malignant glioma Acta radiol 20 (1939) 499
- ILLA P A Cerebral angiography Oxford University Press London 1950
- Lindgren E In Handbuch der Neurochir Bd II Springer Berlin 1954 pp 175 179 180
- LORENZ R Differentialdiagnose der arteriographisch darstellbaren intracranialen Geschwulste Glioblastom Meningeom Sarkom Zbl Neurochir I (1940) 30
- MILLETTI M Die Differentialdiagnose der Gehirngeschwulste durch die Arteriographie Acta neurochir Suppl 1 (1950)
- MONIZ EGAS Die cerebrale Arteriographie und Phlebographie Handbuch der Neurologie Springer Berlin 1940
- SCHIFFER W und TONNIS W Die Bedeutung der Serienangiographie fur die Artdiagnose der Hirngeschwulste Fortschr Rontgenstr 81 (1954) 616
- und LUDWIG G Die Artdiagnose des Meningeoms im Gefasgebild Dtsch Z Nervenheilk 172 (1955) 456
- STATIN S The significance of some angiographic signs of intracranial meningiomas Paper read at the Symp neurorad New York 1964
- WICKBOM I Angiography of the carotid artery Acta radiol Suppl LXXXII (1948) pp 71 77 78 81
- Angiographic determination of tumour pathology Acta radiol 40 (1953) 329
- and STATIN S Roentgen examination of intracranial meningioma Acta radiol 50 (1958) 175
- ZACHARISOV L Angiography of cerebral metastases Acta radiol Diagn I (1963) 521
- ZILCH K J In Handbuch der Neurochirurgie Bd 3 Springer Verlag Berlin Göttingen Heidelberg 1956 pp 399 400 411

changes are often found histologically. Consequently the correct diagnosis is difficult. This is the case in about 20 % of the whole material.

3 If the tumour vessels are observed in the angiogram of an angioblastic meningioma, there is also at least one feeding branch from the external carotid artery to give the diagnosis of a meningioma (not necessarily angioblastic). This is true for about 80 % of the whole material, and emphasizes the necessity of a complete angiographic examination of the vessels, preferably separate injections in the external carotid artery, the internal carotid, and the vertebral artery.

4 If the tumour vessels are irregular in the above mentioned meaning, this is a sign of an angioblastic mode of growth, and the correct diagnosis of angioblastic meningioma is possible, in this material in about 70 % of all the cases (groups IV and III and about half of group II).

5 In order to differentiate the angioblastic meningiomas from other more or less malignant intracranial lesions, it is indispensable to study the circulation through the tumour by means of rapid serial angiography (slow series are not enough). A remarkable fact is the complete absence in this material of rapid arterio-venous shunts (as defined above).

SUMMARY

The angiographic appearance of 46 histologically verified angioblastic meningiomas is described and compared with their histologic structure. The differential diagnosis between ordinary (benign) meningiomas and angioblastic meningiomas as well as between angioblastic meningiomas and other malignant intracranial lesions in general is discussed and emphasis is placed on the value of a complete examination of the external carotid, internal carotid and vertebral arteries and the value of rapid serial angiography as emphasized.

ZUSAMMENFASSUNG

Es wird das Angiogramm von 46 histologisch verifizierten angioblastischen Meningeomen beschrieben und mit ihrer histologischen Struktur verglichen. Es wird die Differenzialdiagnose zwischen den gewöhnlichen (benignen) und angioblastischen Meningeomen wie auch zwischen angioblastischen Meningeomen und anderen malignen intrakraniellen Prozessen allgemein besprochen. Dabei wird auf den Wert einer vollständigen Untersuchung der Carotis ext und int und der Vertebralarterien hingewiesen wie auch der Vorteil rascher Serienangiographie hervorgehoben.

RÉSUMÉ

L'auteur décrit l'aspect angiographique de 46 méningiomes angioblastiques vérifiés histologiquement et le compare à leur structure histologique. Il étudie le diagnostic différentiel entre les méningiomes ordinaires (bénins) et les méningiomes angioblastiques ainsi qu'entre les méningiomes angioblastiques et les autres lésions intracrâniennes malignes en général. Il souligne l'intérêt d'un examen complet des artères carotide externe, carotide interne et vertébrale et l'intérêt de l'angiographie en série rapide.

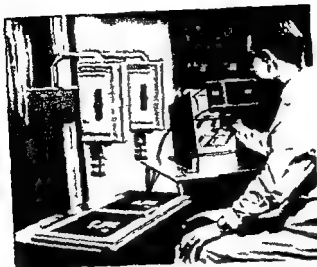


Fig 1 Electronic subtraction equipment. To the left are seen the two televison camera heads and the coordinate table with viewing box for the two films. The electronic amplifier units are placed beneath the viewing box. Monitor and controls for adjusting brightness, contrast and complete registration of the two images are seen to the right. The Polaroid camera is tipped down in front of the monitor.

or intensity of a single detail or of a few details. In its electronic form the difference method (1) is now widely employed in radar for suppressing the stationary background in order to emphasize the radar echoes from moving objects (moving target indication).

If the basic image consists of a conventional roentgenogram and the modification is caused by e.g. the injection of contrast medium into the blood vessels or a body cavity, the method is called subtraction and was presented by ZIEDESS DES PLANTES in his brilliant thesis of 1934.

Characteristic for these difference methods is that

- a) the modified image is reversed in polarity before it is combined with the basic image
- b) the basic image must be stored in some sort of memory before it is combined with the modified image
- c) the procedure requires very high accuracy since the processed image consists of the small difference between two closely alike images
- d) although no new information is in theory created by the difference process, the visibility of the desired details is in certain cases enhanced so considerably that it is in practice permissible to regard the processed image as containing new information.

ELECTRONIC SUBTRACTION

by

HENRY WALLMAN and INGMAR WICKBOM

In the theory of image processing, a family of difference methods has been developed for suppressing unwanted information in an image in order to enhance the visibility of wanted information. These difference methods all have the form

Processed image = basic image minus modification of basic image (1)

If the modification consists of defocusing, the method (1) is called 'contrast equalization', or 'harmonization', or the method of the unsharp mask'. The purpose is to maintain the visibility of contrast differences in small details while suppressing gross variations of contrast from one large area to another. The method is very old in photographic practice, dating from the 1st century, and was applied to roentgenography by SIEGLER in 1930 ('Antidurverfahren'). The 'Logetron' (CRAIG 1954) is an electronic apparatus for achieving harmonization. The connection of one of the authors (H.W.) with image processing arose in 1961 while working on a method for harmonization employing television techniques.

The difference method (1) is used in astronomy, in the study of variable stars (1904), for modifications consisting of a small change in the position

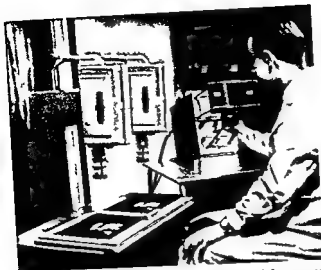


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HENRY WALLMAN and INGMAR WICKBOM

In the theory of image processing, a family of difference methods has been developed for suppressing unwanted information in an image in order to enhance the visibility of wanted information. These difference methods all have the form

Processed image = basic image minus modification of basic image (1)

If the modification consists of defocusing, the method (1) is called 'contrast equalization', or 'harmonization', or 'the method of the unsharp mask'. The purpose is to maintain the visibility of contrast differences in small details while suppressing gross variations of contrast from one large area to another. The method is very old in photographic practice, dating from the last century and was applied to roentgenography by SPIGLER in 1930 ('Antidivulgerföhren'). The 'Logotron' (CRAIG 1954) is an electronic apparatus for achieving harmonization. The connection of one of the authors (H. W.) with image processing arose in 1961 while working on a method for harmonization employing television techniques.

The difference method (1) is used in astronomy, in the study of variable stars (1904), for modifications consisting of a small change in the position

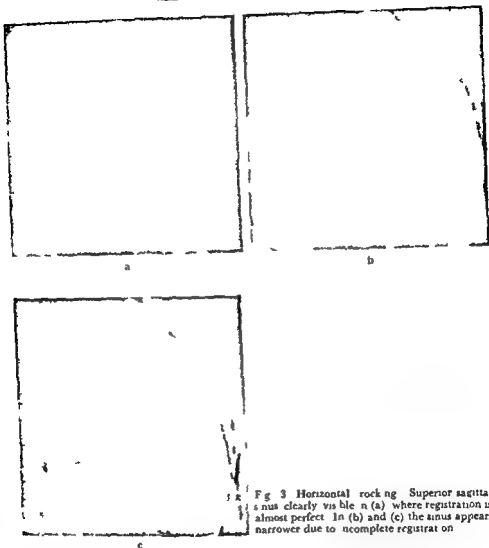


Fig 3 Horizontal rocking Superior sagittal sinus clearly visible in (a) where registration is almost perfect. In (b) and (c) the sinus appears narrower due to incomplete registration.

vertically above the light box. A single synchronizing central contains the sweep generators for the two camera heads and yields a very high level of agreement between the scanning wave forms. Special care is devoted to achieving equality of the input output characteristics of the two camera heads and to maintaining high signal to noise ratios. The camera heads are provided with matched optical systems with variable focal length. Subtraction itself is carried out purely electronically and signal processing after the subtraction

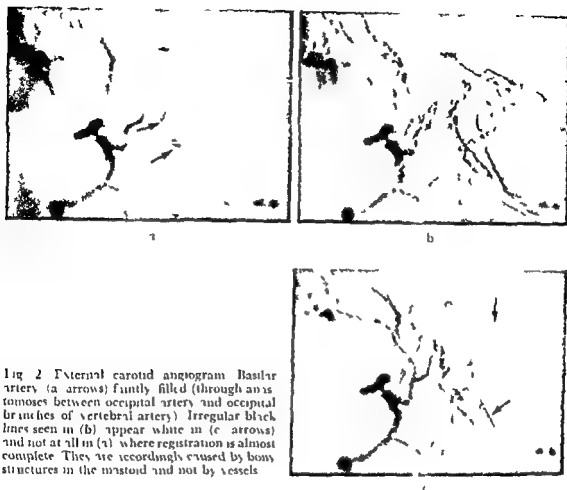


Fig. 2 External carotid angiogram. Basilar artery (a, arrows) faintly filled (through anastomoses between occipital artery and occipital branches of vertebral artery). Irregular black lines seen in (b) appear white in (c, arrows) and not at all in (a) where registration is almost complete. They are accordingly caused by bony structures in the mastoid and not by vessels.

Although these difference methods were all originally devised for photographic methods, the conversion to electronic processing is in each case conceptually simple. Electronic subtraction was proposed and discussed by ZIEDESS DES PLANTES in 1961 and HOLMAN in 1963. The degree of success that can be achieved in electronic subtraction is entirely contingent upon the accuracy of execution of the electronic apparatus, without an exceptionally high standard of registration of the two images and very accurate rendition of the grey scale, the results will be disappointing.

The particular apparatus for electronic subtraction described in this contribution (Fig. 1) was designed with special attention to these aspects of accuracy. It was developed at Chalmers University of Technology, Gothenburg, and has been in use since the autumn of 1963.

The two films to be subtracted are placed on a dual light box with coordinate (x and y axis) movements and two television camera heads are mounted

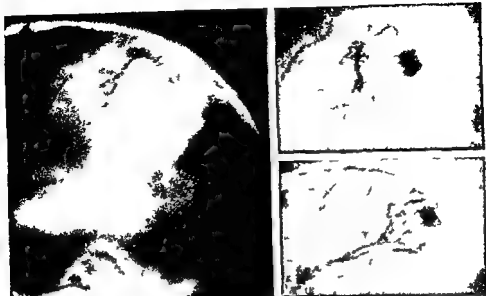


Fig 5 Same case as in fig 4 after injection of external carotid artery (late arterial phase). At top left only one of ascular grooves leading to tumor is visible due to contrast filling of the vein in the other. At right electronic subtraction pictures. At top right the vein mentioned previously is clearly visible while the other groove is subtracted. At centre right where registration is deliberately incomplete the latter is visible as a darker line in upper and a white line in lower part. Tumour staining in poster or part of tumor and draining vein in ante or part are best seen at top right. At bottom right densities are deliberately unbalanced. This case also demonstrates the value of using the subtraction technique when studying the draining veins in meningiomas which in this case are early fillers.

(Figs 2 to 5) while the film base Polaroid images are a step better in grey scale rendition. Neither type of Polaroid material quite reaches the image quality offered by the monitor screen however.

In the photographic subtraction process exact registration of the two images requires a certain degree of manual dexterity; this is unnecessary with the present equipment where fine adjustment is carried out by moving one of the two images electronically in the horizontal and vertical directions rather than mechanically. It is often possible to obtain satisfactory registration in a small area of interest even if the patient has moved slightly between the exposures. If as is not frequently the case doubt arises as to whether a dense line in the image on the monitor is caused by a contrast filled vessel or by

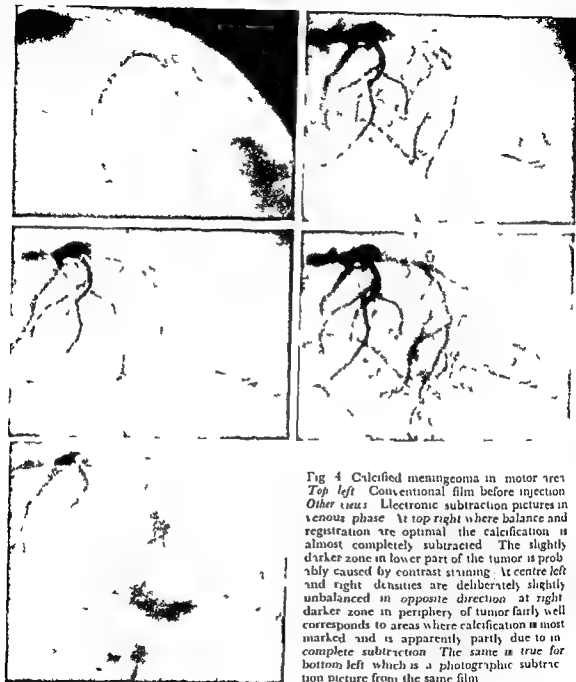


Fig 4 Calcified meningeoma in motor area. Top left Conventional film before injection. Other views Electronic subtraction pictures in venous phase. At top right where balance and registration are optimal the calcification is almost completely subtracted. The slightly darker zone in lower part of the tumor is probably caused by contrast staining. At centre left and right densities are deliberately slightly unbalanced in opposite direction. At right darker zone in periphery of tumor fairly well corresponds to areas where calcification is most marked and is apparently partly due to incomplete subtraction. The same is true for bottom left which is a photographic subtraction picture from the same film.

stage is quite uncritical. The subtraction image is presented on a television monitor with suitable controls for brightness and contrast.

For documentation a Polaroid camera is swung down to photograph the monitor screen. Polaroid positive images on paper are available in ten seconds, and on film base in 60 seconds. Polaroid paper copies are often adequate

ZUSAMMENFASSUNG

Die Autoren berichten über ihre Erfahrungen mit einem neuen elektronischen Subtraktionsapparat. Die elektronische Methode hat gegenüber dem photographischen Verfahren gewisse Vorteile. Die Subtraktionsbilder können unmittelbar nach Entwicklung der Filme erhalten werden. Das Gleichgewicht der Helligkeit und der Kontrast können leicht aufeinander abgestimmt werden, ohne dass eine neue Maske erforderlich ist. Indem man die beiden Bilder ein wenig hin- und herückt, kann auf leichte Weise erkannt werden, ob eine dicke Linie oder ein dichter Fleck in Wirklichkeit einer unvollständig subtrahierten Gefäß- oder Knochenstruktur entspricht. Eine vor dem Bildschirm angebrachte Polaroidkamera erlaubt das Aufnehmen von Bildern zur Archivierung.

RESUME

Les auteurs rendent compte de leur experimentation d'un nouvel appareil de soustraction électronique. La méthode électronique présente certains avantages sur la méthode photographique. Elle permet d'obtenir une image de soustraction immédiatement après le développement du film. On peut facilement régler l'équilibre, la luminosité et le contraste sans avoir à préparer un nouveau masque. En faisant bercer un peu les deux images on juge facilement si une ligne ou une tache dense est vraiment une structure vasculaire ou si elle est une structure osseuse incomplètement soustraite. Un appareil Polaroid placé devant l'écran permet de prendre des images pour les archives.

REFERENCES

- HOLMAN C. B. The application of closed-circuit television in diagnostic radiology. *Proc Mayo Clin* 38 (1964) 67.
 HORENSTEIN R., LUNDH A. and SJOGREN S. E. The subtraction method. *Acta radiol D* 2 (1964) 264.
 ZIESSER DES PLANTES B. G. Planigrafie en Subtractie. Kemink en Zoon, Utrecht 1934.
 — Subtraction. Thieme Verlag, Stuttgart 1961.

an incompletely subtracted bony structure, the question can easily be resolved by 'rocking' one of the images slightly in the vertical or horizontal direction. A skeletal detail then shows up (Figs 2 and 3) with a white edge first on the one side and then on the other, while a contrast filled vessel is always black (provided of course that a positive monitor image is used). Changing the balance can be used for the same purpose.

The use of lenses of variable focal length permits the image to be detail magnified up to four times. Brightness and contrast of the subtracted image can be varied electronically to explore a wide range of densities. The signal levels from the two images can be adjusted individually to achieve balance by varying the diaphragm of one of the cameras, in this way it is possible to accommodate rather large differences in overall density between the conventional film and the angiogram.

Although all these variations can of course also be carried out with the photographic technique, each step would then require a much longer time and often involve processing a new film.

The final results of electronic subtraction can not be better than with a perfect photographic subtraction, assuming the best possible photographic balance, contrast, grey scale and registration for the detail in question. The advantage of the electronic technique is that all the adjustments mentioned can be carried out very quickly, and by the radiologist himself.

Electronics removes subtraction from the photographer and puts it in the hands of the radiologist. In practice this is of great importance. With the photographic method it is not unusual that a subtraction appears to be optimal but is in fact somewhat incomplete in part of the film (Fig 4, bottom left), which may cause error in the interpretation. Such an error can easily be avoided with the electronic method in the manner described above. This is illustrated in Figs 4 and 5, which are also intended to give an idea of the quality of the results obtained.

SUMMARY

The authors report their experiences with a new apparatus for electronic subtraction. The electronic method has certain advantages over the photographic procedure. The subtraction picture can be obtained immediately after the films are processed. Balance, brightness and contrast can be easily adjusted without preparing a new mask. By means of rocking the two images it is easily found out whether a dense line or spot is true vascular or a bony structure incompletely subtracted. A Polaroid camera can be tipped down in front of the monitor allowing pictures to be taken for archivation.

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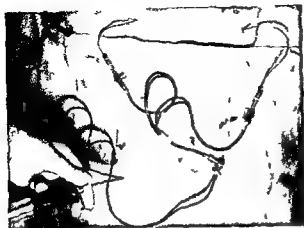


Fig 1 Bilateral percutaneous infraclavicular catheterization of subclavian arteries. Catheter of right and left sides connected to the syringe by a Y shaped tube for a bilateral simultaneous injection

Technique The Seldinger technique for arterial catheterization was adapted to the subclavian artery using the infraclavicular approach (FIELDS & WEIBEL 1964 WEIBEL & FIELDS 1963 WEIBEL et coll 1963). General anesthesia was used in 284 patients and local anesthesia in 130. Bilateral subclavian catheterization was carried out in 371 patients and unilateral catheterization in 43 patients. Once the polyethylene catheter was placed in the subclavian artery, bilateral simultaneous or unilateral selective injection of the vertebral arteries was performed with 12 to 15 ml of Conray 60% being injected in each side (Fig 1).

During the first injection if it was to be unilateral the patient's head and neck were placed in a flat position and turned to the side being injected. For a bilateral simultaneous injection the head and neck were turned to the left side. Following the first injection the position of the head and neck was changed and other projections appropriate to the patient's symptoms or as suggested by the first film were made. The total amount of contrast material injected for a complete study of the vertebrobasilar system varied from 30 to 75 ml for a unilateral injection to 50 to 120 ml for a bilateral study. No single injection exceeded 25 ml of contrast material.

Results

Bilateral simultaneous or unilateral injection of the subclavian arteries has been found to be a satisfactory means for demonstrating the entire vascular tree of the vertebrobasilar system. It readily permits contrast filling of the basilar artery and its branches as obtained by direct injection of the vertebral artery, but the technical difficulties and risk of vertebral artery puncture are

ANGIOGRAPHY OF THE VERTEBROBASILAR ARTERIAL SYSTEM

by

JORGE WEIBEL

Complete demonstration of the vertebrobasilar system has become increasingly important in the diagnosis and determination of proper treatment of lesions involving the cerebral circulation of the posterior fossa. Several techniques employed for this purpose include direct vertebral artery puncture, retrograde injection of the right common carotid, direct infraclavicular or supraclavicular puncture of the subclavian, direct puncture or retrograde catheterization of the brachial or axillary arteries, and injection of the innominate and left subclavian arteries by retrograde femoral artery catheterization. Unfortunately, these techniques do not readily permit bilateral simultaneous or unilateral selective demonstration of the vertebrobasilar system. The purpose of this paper is to report the results of our experience with direct percutaneous infraclavicular catheterization of the subclavian arteries.

This technique was used in 414 patients admitted to the hospital with diagnoses of cerebrovascular insufficiency, intracranial aneurysm, arteriovenous malformation, and brain tumor. Their ages ranged between 11 and 91 years, 285 were male and 129 female.



Fig 3 a) Right subclavian injection. Large normal vertebral and basilar arteries with filling of both posterior cerebral arteries. b) Left subclavian injection with the head and neck turned to the left side. Small left vertebral artery ending in posterior inferior cerebellar artery (lower arrow).

Fig 4 Right posterior oblique view following injection of right subclavian artery in order to avoid superimposition of common carotid artery (upper arrow) over origin of vertebral artery (lower arrow) as seen in fig 10a.

vertebral and basilar arteries and their branches. A difference in size between the right and left vertebral arteries is frequently seen. Often a small vertebral artery does not reach the basilar artery but ends in the posterior inferior cerebellar artery (Fig 3). In some cases one vertebral artery may be absent and the other be of larger caliber. Injection of contrast material into the right

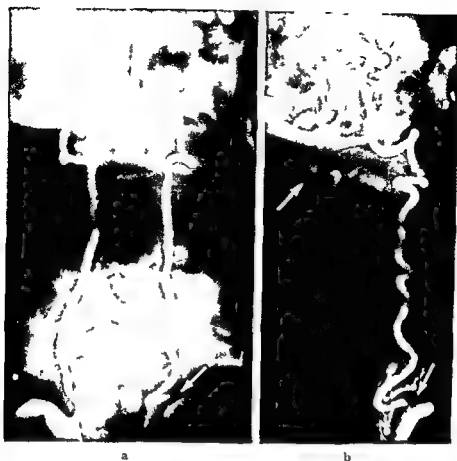


Fig 2 a) Bilateral simultaneous injection of subclavian arteries. Complete contrast filling of vertebrobasilar system. Stenotic lesion and kinking of the left vertebral artery at its origin (arrow). b) Left subclavian injection. Very tortuous vertebral artery with moderate stenosis at its origin (lower arrow). Reverse flow of contrast material into smaller right vertebral artery (upper arrow) and filling of left posterior cerebral artery. (Right posterior cerebral artery filled by right carotid injection.)

eliminated (Fig 2). When a unilateral injection is made, the patient's head and neck are turned to the side of the injection since it has been observed that if the head is turned to the side opposite the injection, the vertebral artery may become blocked at the level of the atlanto axial joint. When bilateral simultaneous subclavian injection is performed, the head is always turned to the left side because the left vertebral artery is usually larger than the right one and a mechanical block can be avoided on the left side at the level of the atlanto axial joint.

By using this method, it is possible for the head and neck to be turned at will, thereby permitting complete demonstration of the entire course of the



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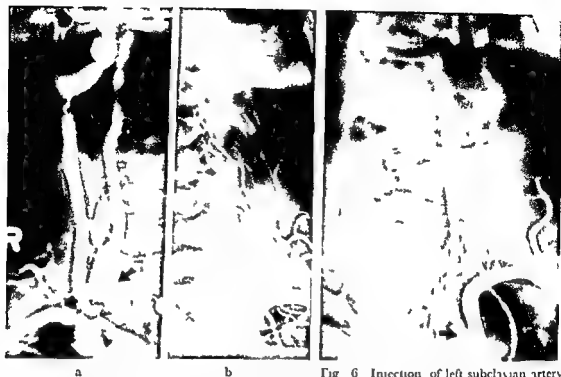


Fig 6 Injection of left subclavian artery with tip of catheter just at its origin. Low origin of left vertebral artery at arch of aorta (arrow)

Fig 5 a) Right subclavian injection with head and neck turned to right side. Tortuous large right vertebral artery with a fusiform aneurysm one centimeter above its origin (arrow). b) Left subclavian injection. Absence of left vertebral artery.

subclavian artery will readily show if the right vertebral artery is absent or occluded at its origin. Sometimes the origin of the right vertebral artery is not demonstrated satisfactorily in a routine anteroposterior view following injection of the right subclavian artery because of superimposition of the right common carotid artery (see Fig 10a). A right posterior oblique view is then necessary to separate the vessels (Fig 4). Great care should be taken in diagnosing complete occlusion or absence of the left vertebral artery since it may originate in the arch of the aorta. In such a case, injection of contrast material must be carried out with the tip of the catheter at the origin of the left subclavian artery. In other cases the vessel may be demonstrated by injection of more concentrated contrast material (Angio Conray 80%) into the aortic arch (Figs 5 and 6).

Arteriosclerotic occlusive disease of the extracranial and intracranial segments of the vertebral artery and occlusive lesions of the basilar artery or its branches are readily demonstrated with this technique (Figs 7 and 8).

Turning of the head and neck to one side in some patients may cause



Fig 7 Right subclavian injection with head and neck turned to side. Moderate stenosis of right vertebral artery at level of sixth cervical vertebra (lower arrow) and severe stenosis at its intracranial segment (upper arrow).

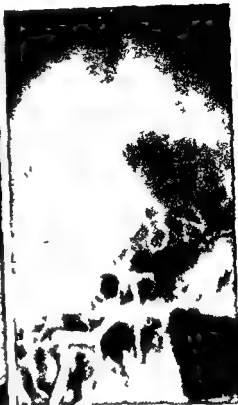


Fig 8 Right subclavian injection. Complete occlusion of left posterior cerebral artery in an anteroposterior position of the head (arrow).

mechanical occlusion of the vertebral arteries in their distal or proximal extracranial segments. Mechanical occlusion in the distal segment at the level of the atlanto-axial joint may occur on the side opposite that to which the head and neck are turned (Fig 9). On the other hand, turning of the head and neck to the side in which the contrast material is injected may block the artery in the proximal segment (Fig 10). In other cases, cervical osteophytes may compress the vertebral artery when the head and neck are turned to one side. To be certain that narrowing or occlusion of the vertebral artery is due to extrinsic compression by musculoskeletal structures, injection must be repeated with the head and neck in the neutral position or turned to the side opposite the mechanical compression. Symptoms of vertebrobasilar insufficiency are enhanced by these positions of the head and neck.



Fig 5 a) Right subclavian injection with head and neck turned to right side. Tortuous large right vertebral artery with a fusiform aneurysm one centimeter above its origin (arrow) b) Left subclavian injection. Absence of left vertebral artery

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Fig 9 a) Bilateral simultaneous injection of subclavian arteries with head and neck turned to right. Complete mechanical occlusion of left vertebral artery at level of atlanto-axial joint (right arrow) and kinking of right vertebral artery at level of second cervical vertebra (left arrow). b) Bilateral simultaneous subclavian artery injection in same patient with head and neck turned to left. The contrast material this time fills the intracranial segment of the left vertebral artery. Degree of kinking of right vertebral artery slightly diminished.

one side. The existence of a small, stenotic or kinked vertebral artery on one side is an important factor in precipitating symptoms when the vertebral artery on the opposite side is partially or completely blocked by turning of the head and neck.

An intrinsic, localized arteriosclerotic lesion in the cervical course of the vertebral artery may simulate an extrinsic compression by a cervical osteophyte. The intrinsic lesion may be readily differentiated because it does not change the form or caliber of the vessel when the head and neck are in different positions.



Fig 10 a) Right subclavian injection with head and neck turned to right side. Mechanical block of right vertebral artery between fifth and sixth cervical vertebrae (arrow). b) Left subclavian injection with head and neck turned to left side. Mechanical block of left vertebral artery at its origin (arrow). c) Bilateral subclavian simultaneous injection in same patient with head and neck in upright position. Both vertebral arteries satisfactorily filled.

Demonstration of intracranial saccular aneurysms of the vertebral and basilar arteries by simultaneous bilateral injection of the subclavian arteries has been very satisfactory in our experience. In this way lamination of blood flow in the basilar artery is avoided, and the head may be moved at will to assist in demonstration of the neck of the aneurysm (Fig 11).

In cases of arteriovenous malformation injection of contrast material both into the anterior and into the posterior circulation of the brain is of primary importance when surgical treatment is contemplated. In one of our patients with an arteriovenous malformation which filled from the right anterior choroidal artery its main feeding artery was a branch of the right posterior cerebral artery (Fig 12). Postoperative right internal carotid and right subclavian angiograms demonstrated that the malformation after clipping of the right anterior choroidal and posterior cerebral arteries was not filled with contrast material.

Complications. The complications in our series have all been local and minimal. In 34 cases small intramural injections in the subclavian artery have

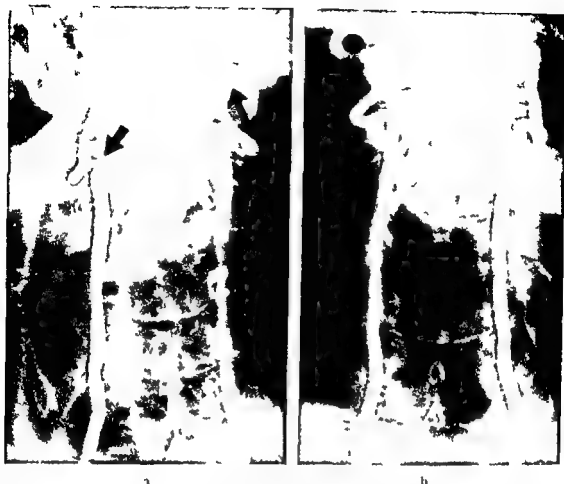


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Fig. 11 Bilateral subclavian simultaneous injection
Aneurysm of bifurcation of brachial artery (arrow)

occurred. Hematoma formation after removing the catheter has been easily prevented by compression of the subclavian artery in both the supraclavicular and infraclavicular regions. Pneumothorax has occurred in 4 cases. Injury of the brachial plexus, arteriovenous fistula, loss of brachial pulse, and cerebral complications have not been observed in this series.

Conclusions

Angiography of the vertebrobasilar system by unilateral or bilateral simultaneous injection through infraclavicular catheterization of the subclavian artery has the following advantages:

1. It is easy to carry out in a relatively short period of time.



Fig. 12 Lateral view of head following injection of right subclavian artery. Arterio-venous malformation feeding from right posterior cerebral artery.

2 It can be performed under local anesthesia in cooperative and alert patients

3 It provides satisfactory demonstration of abnormalities of the entire vertebrobasilar system and its terminal branches

4 It permits the head and neck to be turned in order to study mechanical occlusion of the vertebral artery caused by musculoskeletal structures

5 It provides a means for studying the extracranial and intracranial collateral circulation through the vertebrobasilar system in cases of occlusion of the internal carotid artery

6 It permits injection of contrast material into the lumen of the aortic arch for demonstration of the left vertebral artery when it has an anomalous origin from the arch itself

7 The complications have been local and minimal

SUMMARY

Direct percutaneous infraclavicular catheterization of the subclavian arteries for demonstration of the posterior cervico-cranial circulation is reported in a series of 414 patients. Unilateral or bilateral simultaneous injection proved to be satisfactory for various types of abnormalities of the vertebrobasilar arterial system without the necessity of resorting to direct puncture of the vertebral arteries.

ZUSAMMENFASSUNG

An einer Serie von 414 Fällen wurde die perkutane infraclaviculare Katheterisierung der Art. subclavialis zur Aufzeichnung der hinteren Gehirngefäße durchgeführt. Einseitige oder doppelseitige Simultanfärbung erwiesen sich als nützlich, so dass auf direkte Punktion der Vertebralarterien verzichtet werden konnte.

RESUMÉ

Présentation du cathétérisme sous claviculaire percutané direct des artères sous clavières sur une série de 414 sujets pour mettre en évidence la circulation cervico crânienne postérieure. L'injection unilatérale ou bilatérale simultanée ont donné satisfaction pour divers types d'anomalies du système artériel vertébro basilaire sans qu'il soit nécessaire de ponctionner directement les artères vertébrales.

REFERENCES

- FIELDS W. S. and WEIBEL J. Effects of vascular disorders on the vestibular system. In Neurological aspects of auditory and vestibular disorders. W. S. Fields and B. R. Alford eds. Springfield: Charles C. Thomas (1964).
- — Síntomas vertiginosos producidos por compresión extrínseca de las arterias vertebrales. Acta Neurol. Lit. Amer. 9 (1963) 328.
- WEIBEL J. and FIELDS W. S. Direct percutaneous infraclavicular catheterization of the subclavian artery. J. Neurosurg. 20 (1963) 233.
- — BREITMAN M. E., CRAWFORD E. S. and KIRCHHOFF P. G. Panarteriographie der intra- und extrakraniellen Zirkulation (Ergebnisse mit perkutaner Subclavia Katheterisation). Fortschr. Röntgenstr. 99 (1963) 304.
- — CRAWFORD E. S., DEBAKEY M. E. and BRALL A. C. Jr. Clinical evaluation of Contrast angiography in patients with cerebrovascular disease. Amer. J. Roentgenol. 90 (1963) 1281.
- WEIBEL J. and FIELDS W. S. Angiografía de las afecciones vasculares cerebrales por cateterización infraclaviculaur de la arteria subclavia. In Cirugía Vascular. Sosa Gallardo ed. Barcelona: Miral (in press).

ARTERIES OF THE BASAL GANGLIA

by

GUNNAR WESTBERG

Recent developments in medicine are making an increasing number of intracranial diseases available for therapy without the need for craniotomy. The treatment of certain types of intracranial tumor by high dose supervoltage radiation with rotation (KRAMER, McHISOCK & COLEMAN 1961), the implantation of radioactive substances in cystic tumours (LEAKELL & LIDEN 1951, LINDGREN & WESTBERG 1964) and the treatment of malignant tumors with cytostatic agents are examples of these new possibilities. Furthermore with the increasing use of hypothermia at operations, more advanced neurosurgical interventions have also become possible. The demands for exactitude at the neuroradiologic examination both with respect to the nature and the extent of the pathologic process have become more stringent as a result of these developments. It is often of vital importance to know how a process is related to the basal and central structures of the brain. Such information can seldom be obtained without an angiographic examination and close scrutiny of the ganglionic vessels.

Normal vascular anatomy of the basal ganglia

The investigation reported in this paper is based on the study of 50 corrosion specimens, 100 normal cerebral angiographies and 20 angiographies on cadavers.

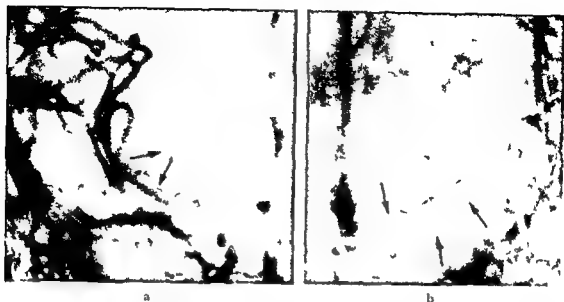


Fig. 1. Anatomical variations. a) The ganglionic arteries from the middle cerebral artery are dominant (→). b) The ganglionic arteries (Heubner's artery) from the anterior cerebral artery are dominant (→). The bifurcation site under the anterior perforated substance is concealed by the middle cerebral artery. In an earlier phase the branches appeared to spring directly from that artery.

ANDERSEN in a paper from 1958 describing the normal anatomy of the lenticulo striate arteries as observed by him at cerebral angiographies, found that with few exceptions these vessels commence 'from the medial part of the middle cerebral artery'. He stressed their value in the angiographic diagnosis of atrophic and space occupying processes. In a later paper in 1963, he described in more detail the appearance of the ganglionic vessels in intracerebral hemioma.

The present author, in a study (1963) comparing the appearance of these vessels in roentgen films and autopsy specimens, reached a different conclusion concerning the normal anatomy. Many of the arterial branches which at the roentgen examination had seemed to start from the middle cerebral artery proved to be branches of Heubner's artery. Others arose from the lateral part of the middle cerebral artery, starting with one to three main branches which ran close to the mother artery in a retrograde direction before curving off laterally to ramify on a level with the bifurcation, or immediately lateral to it. Consequently, these branches in some cases also appeared to arise directly from the middle cerebral artery, as the main branch of the ganglionic vessels was often concealed by that artery in the projections commonly used for angiography. The vessels of the ganglia are not always correctly shown in modern textbooks of anatomy and neuroradiology either. They are often



Fig 2 Normal subject. As both the posterior communicating arteries are filled with contrast medium both the anterior thalamo-perforating arteries (\rightarrow) are visible. The posterior thalamo-perforating arteries (\leftarrow) are visible behind them.

described as consisting of a medial and a lateral group springing from the middle cerebral artery. The anatomist SHELLSHEAR in 1920 demonstrated that the medial part of the anterior perforated substance consists of perforations from a recurrent vessel originating in the anterior cerebral artery in the neighbourhood of the anterior communicating artery. This recurrent vessel seems to be identical with the artery of Heubner. The lateral perforations proved to have been caused by branches from the middle cerebral artery. SHELLSHEAR also found a little group of small arteries situated more laterally than the commonly named lateral striate arteries and penetrating the base of the brain in the *himen insulae* to reach the claustrum. He called these the claustral arteries and this observation is in agreement with my own findings in corrosion specimens of the brain arteries: they are also occasionally seen in carotid angiograms under optimal conditions.

Accordingly if the term 'lenticulostriate arteries' is to be retained it should be remembered that those vessels which usually are called the middle lenticulostriate arteries in the angiographic literature mainly consist of branches from Heubner's artery and thus arise from the anterior cerebral artery; the lateral lenticulostriate arteries on the other hand mainly consist of branches from the middle cerebral artery. To a certain extent a state of balance exists between the distribution of the branches from Heubner's artery and the ganglionic vessels from the middle cerebral artery if the former artery is small the branches from the middle cerebral artery predominate in number and

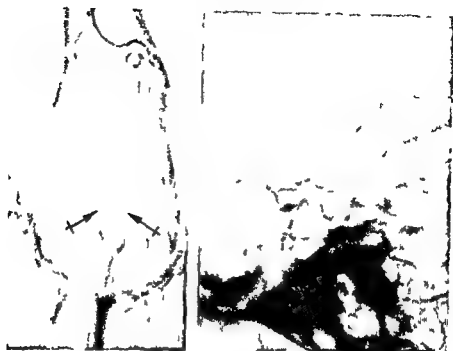


Fig 3 Hydrocephalus. The posterior thalamo perforating arteries are elongated and are visible in the *p* projection (→→)

area of distribution (Fig 1a), whereas if the latter are few, and small, the branches from Heubner's artery are more prominent (Fig 1b). In the study of the corrosion specimens, branches from Heubner's artery often predominated without there being any corresponding signs of this in carotid angiography.

The ganglionic vessels arising from the anterior and middle cerebral arteries are best demonstrated by serial angiography using the *ap* projection with the beam parallel to the floor of the anterior cranial fossa, the most suitable projection may vary slightly in the individual case, however. The main stem of Heubner's artery, even when narrow, is usually well seen in the oblique projection. In the lateral view, the ganglionic vessels have a fan-like appearance.

A number of small perforating arteries arise from the posterior communicating and posterior cerebral arteries and supply the thalamus and adjoining structures. The naming of these vessels has also caused some confusion in the anatomical and radiologic literature, names such as pre-mammillary or thalamo-tuberal, retro-mammillary or thalamo-perforating, and thalamo-geniculate arteries, are commonly encountered.

Several small branches spring from the posterior communicating artery to run toward the base of the brain. As a rule, one of these is larger than the others and is nearly always seen in the lateral projection, at rapid serial



Fig 4 Hydrocephalus. The ganglionic arteries are displaced laterally (\rightarrow)

angiography when the posterior communicating artery is filled with contrast medium. Usually it is best seen at vertebral angiography (Fig 2) but some times it is visible at carotid angiography. The writer calls this large vessel arising from the posterior communicating artery the anterior thalamo perforating artery. Normally it runs obliquely upwards and backwards and penetrates into the base of the brain lateral to the mamillary body, it ramifies within or immediately inside the lateral wall of the third ventricle and may be followed up to the level of the massa intermedia. The anterior thalamo perforating artery supplies the outer anterior part of the thalamus.

The posterior thalamo perforating arteries spring from the posterior cerebral artery. Their appearance is that of a bunch of small arteries running in the interpeduncular fossa in the same direction as the anterior perforating artery. They originate between the posterior communicating and basilar arteries perforating the base of the brain behind the mamillary bodies in the posterior perforated substance (Fig 2) and supplying among other things, the posterior portions of the thalamus. Sometimes they spring almost entirely from one side but in many cases they arise from the posterior cerebral artery on both sides. Some of the posterior twigs run to the mesencephalon. The posterior thalamo perforating arteries are also best seen in the lateral view in the ordinary a p half axial projection they are sometimes hidden by the

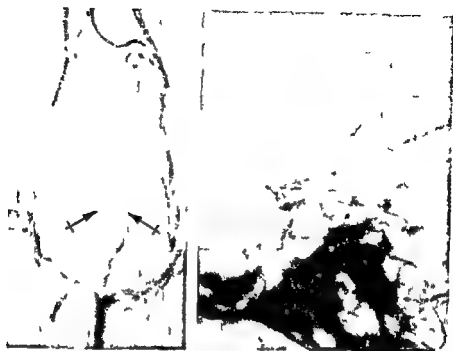


Fig 3 Hydrocephalus The posterior thalamo perforating arteries are elongated and are visible in the a.p. projection (→)

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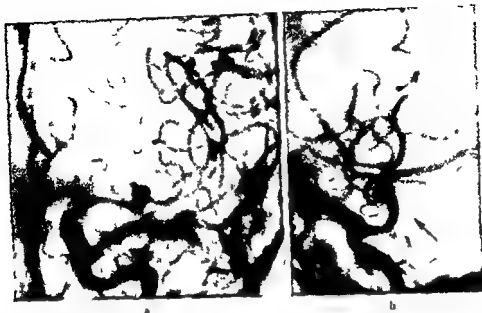


Fig 6 a) Arterial aneurysm of the middle cerebral artery. The ganglionic arteries which are duplicated locally arise close to the aneurysm. b) The anterior thalamo-perforating artery springs from the top of an infundibular widening (→)

most lateral of the ganglionic vessels has therefore proved to be a better point of reference for measurement of the distance to the midline (in the following called the ganglionic vessel distance) and this principle was followed exclusively in the measurements undertaken in the present material. Determinations were carried out *in vivo* on 72 hemispheres which were also examined by encephalography: normal subjects and cases with symmetric dilatation of the ventricular system were studied. The width of the lateral ventricles was assessed at encephalography by measuring the shortest distance from the inner upper angle of the lateral ventricle to the caudate nucleus in the a.p. pneumogram as well as the greatest transversal diameter in the same projection. The former measure which according to KJELDSEN (1958) gives a better indication of ventricular size than the transversal diameter was used by GALLOWAY, GREITZ & SJÖGREN (1964) when comparing the degree of ventricular dilatation with the distance between the lateral choroidal and posterior pericallosal arteries in the lateral projection at vertebral angiography. The author's measurements are shown in Fig 5. It emerges clearly from these diagrams that whatever the method used for calculation of ventricular size at encephalography, there is a definite correlation between ventricular size and the ganglionic vessel distance. According

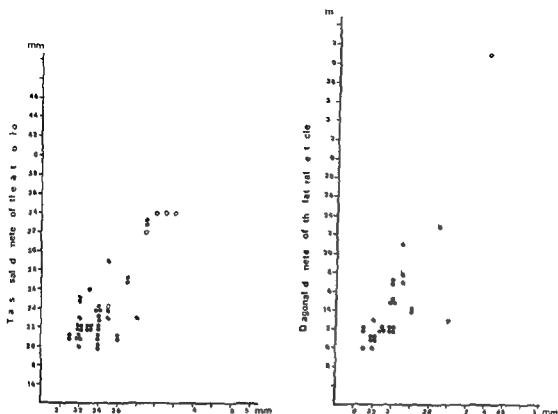


Fig. 4. Measurements showing ganglionic vessel distance. White circles denote cases with obstructive hydrocephalus.

vermis branches of the posterior inferior cerebellar artery and are then inaccessible for study. They are sometimes visible, for instance, in cases of hydrocephalus (Fig. 3).

The basal ganglia are also supplied with blood by small branches running from the anterior and posterior choroidal arteries.

Pathologic conditions

Dilatation of the lateral ventricles may be diagnosed by studying the ganglionic vessels, which are capable of being displaced laterally (Fig. 4). ANDERSEN (1958) measured the distance from the lenticulo striate arteries to the midline in normal patients and in cases with central and cortical atrophy, taking 'distance from midline of skull to most lateral point on the convexity of the curve of the most medial and the most lateral of the ventriculo striate arteries lying in the arterial fan'. The present author has found that the distance from the most medial of the arteries to the midline is difficult to assess, since the medial ganglionic vessels gradually decrease in size as they approach the midline, the

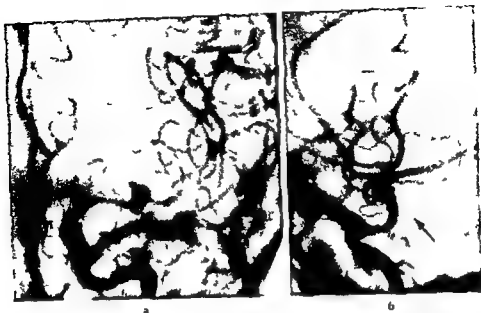


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Fig 7 Two arteriovenous malformations supplied only through the ganglionic arteries a) Heubner's artery Subtraction film b) The posterior thalamo perforating arteries



Fig 8 An arteriovenous malformation partly supplied through a greatly dilated ganglionic artery from the middle cerebral artery



Fig 9 Intracerebral hemorrhage a) Postmortem angiography with a water soluble contrast medium. The medium has leaked from the ganglionic arteries to the hematoma (→) b) A specimen from the same case. The Sylvian fissure is markedly displaced in a lateral direction

to these calculations the ganglionic vessel distance agrees better with the greatest transversal diameter of the anterior horn than with the diagonal diameter. The greater the size of the lateral ventricles the better is the correlation.

Vascular disease. A knowledge of the special anatomy of the basal ganglionic arteries is important in operations for arterial aneurysm. As the origin of these vessels often lies close to aneurysms on the main brain arteries they run a considerable risk of being damaged at the operation with grave neurologic deficits as a result. The operative risks are increased if the ganglionic vessels are seen at angiography to be situated near the aneurysm (Fig 6a). When infundibular widening is present a narrow little artery is sometimes seen running from its apex obliquely and backwards. This is usually the anterior perforate artery (Fig 6b). If an infundibulum is considered to be due to an aneurysm and is treated surgically there is a risk that trophic damage will arise in the basal ganglia or the thalamus. Small intracerebral hemorrhages which are common occurrences in association with subarachnoid bleeding, often appear as a local displacement at the level of the aneurysm (Fig 6a).



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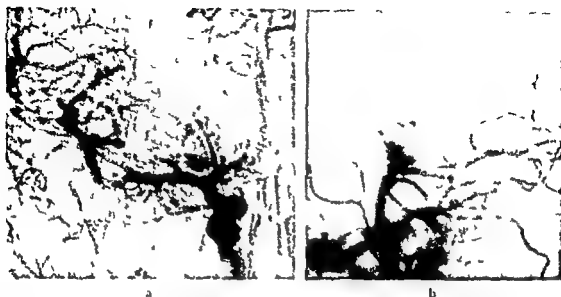


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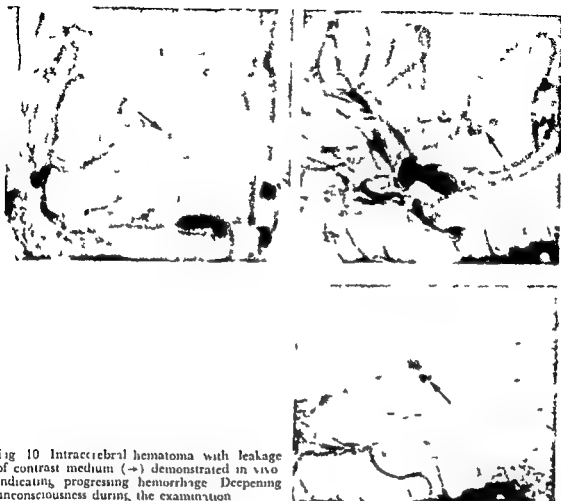


Fig 10 Intracerebral hematoma with leakage of contrast medium (→) demonstrated *in vivo* indicating progressing hemorrhage Deepening unconsciousness during the examination

Arteriovenous malformations involve the arteries of the basal ganglia both directly and indirectly via collaterals. An arteriovenous malformation in which Heubner's artery was the only afferent vessel is shown in Fig 7a, while in Fig 7b is seen a malformation supplied by the posterior thalamo perforating arteries. Because of the risk of central necrosis neither of these patients were operated upon.

Expanding lesions The appearance of the ganglionic vessels often allows fairly accurate assessment of the location and extension of expansive processes in and near the basal ganglia. This is especially true in the case of central and temporal lesions, but a study of these small arteries often gives information of value about processes in the parietal and frontal regions. ANDERSEN has discussed the displacement of the ganglionic vessels in intracerebral hemorrhage. He pointed out that the small ganglionic arteries may show considerable

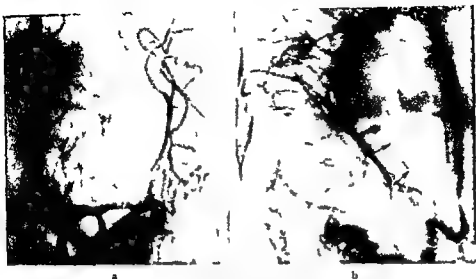


Fig 11 Temporal expansive processes a) Intracerebral tumor infiltrating the basal ganglia. The distance between the ganglionic and the Sylvian arteries is increased and the latter vessels are displaced medially b) Extracerebral tumor. Both Sylvian and ganglionic arteries displaced medially and distance between them decreased



Fig 12 Parietal astrocytoma. The ganglionic arteries are displaced basally and medially

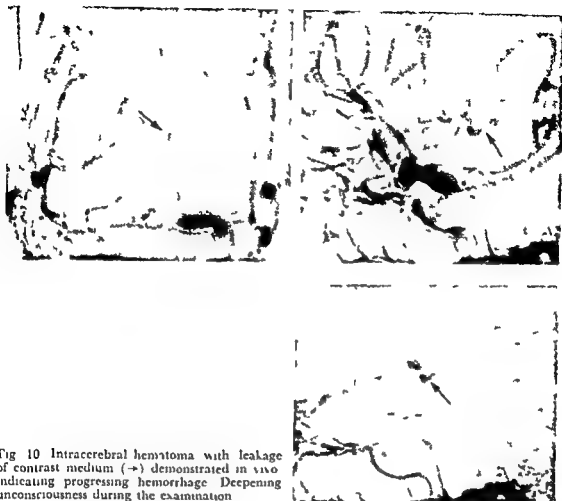


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Fig 14 Upward herniation. The anterior and posterior (lalamo-perforating) arteries are elongated and displaced in a forward direction.

apart. An increase of the distance between the Sylvian and the ganglionic vessels in infiltrating temporal and central tumors is usually a sign that the tumor is growing in both a temporal and a central direction in the basal ganglia (Fig 11a). A similar increase in this distance may also be seen in other conditions; however, it is seen for example as a side phenomenon of lateral herniation of the cerebral hemisphere under the falx in cases with large frontal and occipital processes. The distance is also increased in basal, extracerebral tumors since the tumor can displace the insula region medially and adjoining parts of the temporal lobe laterally. A decreased or unchanged distance between the Sylvian and the ganglionic vessels in conjunction with medial displacement of both these groups of vessels is often seen in cases with extracerebral processes in the anterior and lateral part of the temporal region at the level of the basal ganglia. This type of dislocation is seen for example, in a hygroma (Fig 11b). The latter condition if located in the anterior and lateral temporal region is sometimes difficult to distinguish in an ordinary *ap* view. If the ganglionic vessels arising from the anterior and middle cerebral arteries as well as the Sylvian arteries are displaced medially and the distance between the two groups of arteries is not increased then the presence of a laterally situated extracerebral process should be suspected and suitable oblique views taken. Similarly, if an extracerebral hematoma has been diagnosed, intracerebral hemorrhage in the temporal lobe should be suspected if the distance between the Sylvian and the ganglionic vessels is increased. If an



Fig. 13 Thalamic tumor a) Vertebral angiography. The thalamo-perforating arteries are elongated and the anterior thalamo-perforating artery (→) leaves the posterior communicating artery at right angles b) Carotid angiography. The elongated and displaced anterior thalamo-perforating artery is also seen at carotid angiography. This was the only sign of a tumor in the thalamus at this examination.

displacement even in cases where the superficial vessels are not noticeably out of position. He also noted that hemorrhages which do not perforate into the ventricular system may cause a fair degree of displacement even if they are small, with regard to hemorrhages perforating into the ventricular system, he stated that "the displacement of the pericallosal and/or middle cerebral arteries is insignificant and within normal variations". The present author does not entirely share his opinion. True, the pericallosal artery usually is then situated in the midline, or is only slightly displaced, but the middle cerebral arteries as a rule are curved and elongated, and displaced laterally towards the calotte. Fig. 9, a and b shows a case in point, in which the angiography had been carried out post mortem and the injected contrast medium had leaked out into the hematoma. Leakage of contrast substance into the hematoma may also occur in vivo (Fig. 10).

The ganglionic arteries often show characteristic changes in different forms of intracerebral and extracerebral expansive processes. A tumour which has originated in the basal ganglia and has not yet become especially large may manifest itself solely as a deformation and pushing apart of the basal ganglionic arteries without any other signs of dislocation of the larger vessels, if it has also infiltrated into the temporal lobe the Sylvian vessels as well are forced



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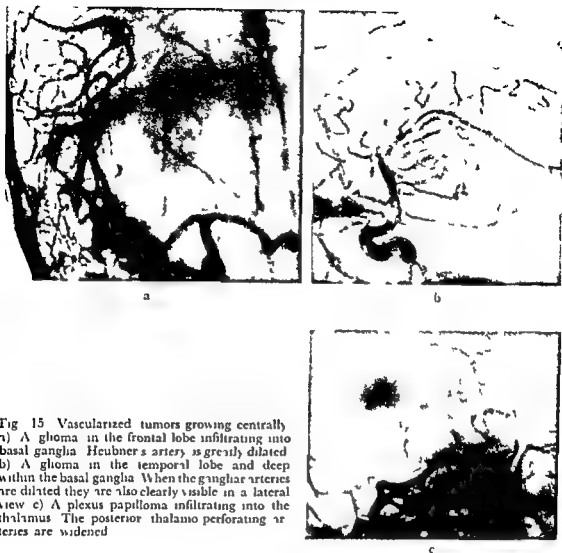


Fig 15 Vascularized tumors growing centrally
 a) A glioma in the frontal lobe infiltrating into basal ganglia Heubner's artery is greatly dilated
 b) A glioma in the temporal lobe and deep within the basal ganglia When the ganglionic arteries are dilated they are also clearly visible in a lateral view
 c) A plexus papilloma infiltrating into the thalamus The posterior thalamo perforating arteries are widened

expanding process is present parietally the ganglionic arteries will be compressed and displaced basally and medially (Fig 12)

Central tumors growing in the thalamus region cause a characteristic elongation and displacement of the thalamo perforating arteries, and in such cases the angle between the anterior thalamo perforating and posterior communicating arteries, which normally is sharp and opens backwards, is often widened (Fig 13, a and b) The anterior and posterior thalamo perforating arteries may even both point anteriorly A similar change in direction may also be seen in upward herniation through the tentorial notch in cases with expanding processes in the posterior cranial fossa (Fig 14) Although the anterior thalamo perforating artery is very small, it is of considerable

importance in the diagnosis of processes in the thalamus region. At a carotid angiography with apparently normal findings, an elongation of this vessel in conjunction with enlargement of the angle with the posterior communicating artery may be the only sign but a very clear one, of a pathologic process in the thalamus (Fig. 13b).

In highly vascularized tumors the ganglionic vessels become increasingly dilated as the tumor infiltrates into the central and basal parts of the brain. Widening of Heubner's artery may be an indication that the tumor is also growing into the anterior part of the basal ganglia and internal capsule (Fig. 15a). If the tumor is located a little farther back and higher up the ganglionic vessels from the middle cerebral artery will also be dilated and deformed (Fig. 15b) and if it is in the thalamus region the posterior thalamoperforating arteries are dilated (Fig. 15c).

SUMMARY

The normal roentgenographic anatomy of the arteries of the basal ganglia and their appearance in different pathologic conditions in the brain are described. It is of value to study not only the ganglionic vessels springing from the anterior cerebral and middle cerebral arteries but also those which arise from the posterior communicating and posterior cerebral arteries.

ZUSAMMENFASSUNG

Es wird die normale Röntgenanatomie der Arterien der basalen Ganglien und ihr Aussehen bei verschiedenen pathologischen Zuständen beschrieben. Es ist wichtig nicht nur die Gefäße die von der art. cerebr. ant. und media sondern auch die die von der art. communicans post. und von den art. cerebr. post. entspringen zu studieren.

RÉSUMÉ

Description de l'anatomie radiologique normale des artères des noyaux gris centraux et de leur aspect dans diverses affections cérébrales. Il importe d'étudier non seulement les artères des noyaux gris qui proviennent des artères cérébrales antérieure et moyenne mais aussi celles qui viennent des artères communicante postérieure et cérébrale postérieure.

REFERENCES

- ANDERSEN POLL E. The lenticulo-striate arteries and their diagnostic value. A preliminary report. Acta radiol. 50 (1958) 84.
 — Angiographic localization of small intracerebral hematomas. Acta radiol. New series D. agnos. 1 (1963) 173.
 CRITCHLEY MACDONALD. The anterior cerebral artery and its syndromes. Brain 53 (1930) 190.

- KNUDSEN, P. A. Ventriklernes størrelsesforhold i anatomisk normale hjerner fra voksne mennesker (Danish) Andersbogtrykkeriet, Odense 1958
- GALLOWAY J. R. GREITZ I. and SJOGREN S. E. Vertebral angiography in the diagnosis of ventricular dilatation. *Acta radiol. New Series. Diagnosis* 2 (1964) 321
- KRAMER, S. McKISSOCK W. and CONCANNON J. P. Craniopharyngeomas. Treatment by combined surgery and radiation therapy. *J. Neurosurg.* 18 (1961) 217
- LEKSELL L. and LIDEN K. A therapeutic trial with radioactive isotopes in cystic brain tumor. In: *Radioisotope Techniques Vol. I Medical and physiological applications* (= *Proc. Isotope Techniques Confer.* Oxford July 1951) H. M. Stationery Office, London 1951
- LINDGREN E. and WESTBERG G. Radioactive bismuth phosphate for the treatment of craniopharyngioma. *Acta radiol. New Series Therapy physics biology* 2 (1964) 113
- WESTBERG G. The recurrent artery of Heubner and the arteries of the central ganglia. *Acta radiol. New series Diagnosis* 1 (1963) 949

DAS CAROTISANGIOGRAMM BEI DEN EINSEITIG FRÜHKINDLICHEN HIRNSCHADIGUNGEN

VON

HEINZGEORG VOGELANG

Die Diagnose einer Hemiatrophia cerebri gewinnt bei dem heutigen Stande der Neurochirurgie zunehmend an Bedeutung. Maßnahmen wie die Hemisphärektomie vermögen das meist mit der spastischen Halbseitenlähmung vergesellschaftete Anfallsleiden und die dadurch bedingte zunehmende Wesensänderung zum Stillstehen zu bringen. In vielen Fällen gelingt dann ein Aufholen des geistigen Entwicklungsrückstandes und eine soziale Eingliederung.

Wenn auch heute noch die Encephalographie unter den diagnostischen Maßnahmen an erster Stelle steht, da sie u. a. eine Aussage über die vom Operateur geforderte halbseitige Schädigung gestattet, so hat doch die cerebrale Angiographie den Vorteil, in vielen Fällen die Ursache der Schädigung aufzudecken und somit einen Beitrag zur Pathogenese zu leisten. Die zunehmende technische Verbesserung der Serienangiographie und der Narkose gestatten es heute ohne erhöhtes Risiko auch Kleinkinder und Säuglinge dieser Untersuchung zu unterziehen und so schon frühzeitig gefäßabhängige cerebrale Schädigungen festzustellen. Derartige Gehirnschädigungen im Kindesalter sind seit den grundlegenden pathologisch-anatomischen Arbeiten

Tabelle

Übersicht der erhobenen angiographischen Befunde (unter Berücksichtigung des Entstehungszeitpunktes)

Gesamtzahl der Angiogramme (36)

Verschluss der A cerebri med	(6)	}	pranatal	(10)
Teilausfall der A cerebri med	(5)		natal	(0)
Dunkkalibrige Arterien	(6)		postnatal	(7)
Arachnoidalzysten	(3)			
Verziehung von Art zur atrophischen Seite	(9)	}	pranatal	(0)
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Da sich unsere Klinik unter anderem speziell mit den Problemen der infantilen Hemiplegie (LANG PIA) und deren operative Behandlung durch die Hemisphärektomie (PIA) befasst, lag es nahe unser eigenes Material mit der Frage der Wertigkeit der Carotisangiographie und ihrer Ergebnisse zu überprüfen.

Es wurden 36 Angiogramme ausgewertet (20 weibliche und 16 männliche Pat. im Alter zwischen 4 Wochen und 35 Jahren). 17 Male war die linke, 19 Male die rechte Grosshirnhemisphäre die geschädigte. 16 der Fälle zeigten entweder einen ganz unauffälligen Befund (7 Fälle) oder nur eine Verziehung von Arterien und Venen zur atrophischen Seite hin (9 Fälle). Dreimal fand sich eine Arachnoidalzyste. Bei den übrigen 17 Angiogrammen fanden sich Veränderungen an den Arterien selbst und zwar in 6 Fällen ein kompletter Verschluss der A cerebri med (zweimal als Aplasie imponierend) in 5 Fällen ein Ausfall grosserer Äste bzw. Gruppen von Nebenästen dieser Hauptarterie und weitere 6 Fälle zeigten auffällig dunkelkalibrig = T wie rarefiziert.



Abb 1 Hemiatrophie cerebri Ausfall der A. cerebri med

wirkende Arterien der Mediagruppe. Nur in einem Fall fand sich neben einem Ausfall von Arterien des Mediabereiches mit Kollateralkreislaufen auch ein Fehlen oberflächlicher Hirnvenen fronto parietal und Nichtdarstellung des Sinus sag. sup. in seinem vorderen Anteil.

Diese angiographischen Befunde bedürfen aber in mehrfacher Hinsicht noch weiterer Erörterungen. Da ist einmal der Zeitpunkt der Entstehung derartiger Gefäßveränderungen. Unter den 17 Fällen mit pathologischen Angiogrammen war die Schädigung in 10 als in der Pränatalperiode und in 7 Fällen als postnatal entstanden anzusehen. Kein Fall ist dabei der mit Überzeugung in die Geburtsperiode zu verlegen ist. Dem gegenüber stehen unter den Fällen bei denen die Angiographie normal ausfiel bzw. nur eine Verziehung von Gefäßen zur Gegenseite aufwies in 4 Fällen eine Schädigung unter der Geburt und in 12 Fällen postnatal, jedoch keiner im Pränatalzeitraum. Diese Auffälligkeiten stimmen mit pathologisch anatomischen Ergebnissen überein, nach denen geburtsbedingte Früh- und Spätschädigungen vorwiegend doppelseitige Ausfälle hervorrufen und die arterielle Gefäßschädigung als Ursache der infantilen Hemiplegie vorwiegend pränatal entsteht. Auf Grund seiner Erfahrung an inzwischen 80 ausgewerteten infantilen Hemiplegien neigt PIA auch dazu eine pränatale Entstehung bei überwiegend schleichendem und negativen Verlaufsförmigen mit Fehlen jeglicher atologischer Hinweise anzunehmen. Unsere gefäßabhängigen Schädigungen zeigten nahezu alle einen derartigen Verlauf. Als Ursachen derartiger Gefäßschädigungen haben Embolien bei Infektionskrankheiten, sonstigen Infekten oder Herzerkrankungen (BANKER, FORD, MAIR, WIESEL u. a.) zu gelten. Ferner lokale oder allgemeine Gefäßerkrankungen mit Thrombosen, Degenerationen, Fibroblastosen (BANKER, FIELD, STEVENS, WISOFF

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Abb 3 Hemiatrophia cerebri. Auffallend dunndkalibrige Arterien der gesamten Mediagruppe (a) im Vergleich mit der gesunden Seite (b) Congenital oder Rekanalisiert

Die drei angeführten Arachnoidalzysten bedurften keiner besonderen Überlegung. PIA wies 1963 erstmals auf Arachnoidalzysten als Ursache infantiler Hemiplegien hin. Die angiographischen Veränderungen sind durch den raumfordernden und die Art des Prozesses bedingt eine pränatale Entstehung ist anzunehmen. Ob bei den als normal bezeichneten Angiogrammen bzw. denjenigen die nur eine Verziehung von Gefäßen zur atrophischen Seite hin aufwiesen die Schädigung etwa in der Peripherie der Gefäße gelegen ist und sich somit dem angiographischen Nachweis entzieht muss offen gelassen werden. Dass dies der Fall sein kann ist seit den Arbeiten von EICKE MEYER u. a. bekannt. Da in unseren Fällen jedoch ein pathologisch anatomischer Befund fehlt kann eine Aussage hierzu nicht erfolgen.

ZUSAMMENFASSUNG

Von 36 ausgewerteten Carotisangiographien bei Patienten mit einseitigen frühkindlichen Hirnschädigungen zeigten 17 (47,2 %) Veränderungen an der A. cerebri media und deren Äste. Bei einem Fall fanden sich zusätzlich noch Auffälligkeiten am Venensystem. In der überwiegenden Mehrzahl war die Schädigung in die Intrauterinperiode zu verlegen. Diese Befunde entsprechen sowohl der pathologisch anatomischen Erfahrung als auch der anderer Untersucher.

SUMMARY

The evaluation of carotid angiography in 36 cases of unilateral cerebral damage in young children revealed changes in the medial cerebral artery or its branches in 17 cases (47.2 %). In one case additional abnormalities in the venous system were evident and in most cases prenatal damage could be assumed. The findings correspond well with the views held by neuropathologists and with the experiences of other investigators.

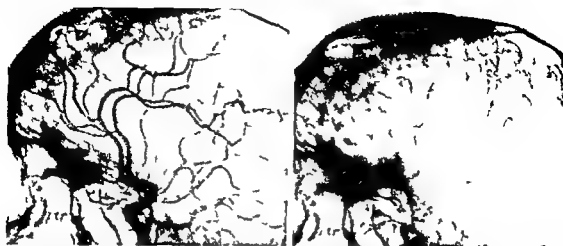


Abb 2 Hemiatrophia cerebri Weigehender Ausfall der Aa ascendentes frontoprecentral und dunnkalibrige Gefässe der Sylviagruppe (Rekanalisiert?)

u r), auch kommen kongenital angelegte Ausfälle, z T verbunden mit anderen Missbildungen (MYRM, NORMAN & URICH, PIA, WOLMAN) vor. Drei der eigenen Fälle wiesen Gefässveränderungen (einmal der Ausfall des Hauptstammes der Sylviagruppe und zweimal dunnkalibrige Gefässe) auf, die im Anschluss an ein schweres Schädellurntrauma im Frühkindesalter entstanden sind.

Eine weitere Frage ist, welche Gefässe von derartigen Schädigungen betroffen sind. Alle Autoren stimmen darin überein, dass in erster Stelle die A cerebri med und ihre Äste stehen, dann folgen mit Abstand die A crotis int, die A cerebri post und ant (BANKER, DYKEN, DUFFY et coll, GOLDSTEIN & BURGESS, LEBEVRE et coll, LANG & GOLDBERG, TONNIS, WILLIE u a). In unserem Material ist es ausschliesslich die mittlere Hirnarterie und deren Äste, eine Veränderung an einer anderen grossen Hirnarterie kam nicht zur Darstellung. Dabei wurden sowohl komplette Verschlüsse — zweimal imponierte der Ausfall der A cerebri med als eine Aphasie —, Teilausfälle und das bekannte Bild der dunnkalibrigen Arterien beobachtet. Bei den letztgenannten Auffälligkeiten (6 Fälle) dürfte es sich zum Teil um Rekanalisierungen handeln, wie dies nicht nur experimentell, pathologisch anatomisch sondern auch angiographisch am Menschen gesichert ist (u a BECKER, DECKER, HEUBNER, MAIR, WOLMAN). Das auch in unserem Material praktische Fehlen rein Venöser Veränderungen als Ursache der Hemiatrophie ist dadurch zu erklären, dass venöse Verschlüsse und Abflussbehinderungen zwar einmal zu rein halbseitigen Ausfällen führen können (LANG), jedoch ausserordentlich selten sind (PIA). Wir selbst beobachteten nur einmal eine Kombination mit arteriellen Veränderungen bei einem Mädchen, welches im Alter von 10 Jahren plötzlich eine Halbseitenlähmung erlitt.

- MYNIN D. Carotid thrombosis in childhood. *Arch Dis Child* 35 (1960) 515
- NORMAN R. M. and URICH H. Dissecting aneurysm of the middle cerebral artery as a cause of acute infantile hemiplegia. *J Path Bact* 73 (1957) 580
- PIA H. W. Die Hemisphärektomie in der Behandlung der cerebralen Kinderlähmung. *Dtsch Z Nervenheilk* 181 (1960) 395
- Die operative Behandlung der infantilen Hemiplegie aus prophylaktischer Sicht. *Prophylaxe u. Therapie* Bd 2 S. 1—2. Wien: Brasec 1963
- Neue Wege zur Behandlung der cerebralen Kinderlähmung. *Tgl Praxis* 1 (1960) 430
- Ätiologie und Pathogenese der infantilen Hemiplegie. *Dtsch Z Nervenheilk* 185 (1963) 357
- PICAZA J. A. Cerebral angiography in children. *J Neurosurg* 9 (1952) 235
- POSER C. M. and TAVERAS J. M. Clinical aspects of cerebral angiography in children. *Pediatrics* 16 (1955) 73
- ROEGAY H. and WELCH K. Peripheral collateral circulation between cerebral arteries. *J Neurosurg* 11 (1954) 363
- SCHWARTZ PH. und FRAU L. Morphologie und Entstehung der geburts-traumatischen Blutungen im Gehirn und Schädel der Neugeborenen. *Z Kinderheilk* 40 (1923) 427
- STEVENS H. Carotid artery occlusion in childhood. *Pediatrics* 23 (1959) 699
- TAVERAS J. M. and POSER C. M. Roentgenologic aspects of cerebral angiography in children. *Amer J Roentgenol* 82 (1959) 371
- TENG F. and GOLDENBERG E. D. Thrombosis of internal carotid artery in a 5-year-old child. *A M A J Dis Child* 99 (1960) 228
- TONNIS W. Zit. nach LANG
- VIGOLROUX R., BONDONRESQUES J., FIGANOL G. et VIGOLROUX M. Valeur respective de la pneumographie, de l'angiographie et de l'électroencephalographie au cours des encéphalopathies infantiles. *Rev Neurol* 90 (1954) 662
- WALTER W. und BRANDT P. Die Angiographie bei den frühkindlichen Hirnschäden. *Acta Neurochir* 6 (1958) 310
- WEISEL J. Über Arterienveränderungen bei kindlichen Infektionskrankheiten. *Wien klin Wochr* 56 (1961) 15
- WISOFF H. S., YOUNKERS Y. and ROTHBALLER A. Cerebral arterial thrombosis in children. *Arch Neurol Psychiat* 86 (1961) 258
- WOLMAN L. Cerebral dissecting aneurysms. *Brain* 82 (1959) 276
- WYLLIE W. G. Acute infantile hemiplegia. *Proc roy Soc Med* 41 (1948) 459

RÉSUMÉ

Sur angiographies carotidiennes de malades présentant une lésion cérébrale unilatérale datant de la première enfance, 17 (47,2 %) ont montré une altération de l'artère cérébrale moyenne et de ses branches. Dans un cas, il y avait en outre des anomalies du système veineux. Dans la grande majorité des cas la lésion pouvait être rapportée à la période prénatale. Ces résultats concordent aussi bien avec les constatations anatomo-pathologiques qu'avec l'expérience d'autres auteurs.

LITERATUR

- ACHSLOGH J. et SAINTES M. J. L'arteriographie normale et pathologique du nourrisson et du jeune enfant. *Acta neurol. belg.* 61 (1961) 102
- BANKER S. Cerebral vascular disease in infancy and childhood. *J. Neuropath. exp. Neurol.* 20 (1961) 127
- BECKER H. Über Hirngefäßausschaltungen. *Dtsch. Z. Nervenheilk.* 161 (1949) 446
- DECKER K. Klinische Neuroradiologie. Georg Thieme Stuttgart 1960
- DUFY P., PORTNOY B., MAURO J. and WEIRLE P. Acute infantile hemiplegia secondary to spontaneous carotid thrombosis. *Neurology* 7 (1957) 664
- DIKEN M. Angiographic study of the middle cerebral artery in chronic infantile hemiplegia. *J. Neurol. Neurosurg. Psychiat.* 27 (1964) 326
- EICKE W. J. Gefäßveränderungen bei Meningitis und ihre Bedeutung für die Pathogenese frühkindlicher Hirnschaden. *Virchows Arch. path. Anat.* 314 (1947) 88
- FIELD. Zit. nach PIA
- FORD F. R. Disorders of the nervous system in infancy, childhood and adolescence. 4. Ed. Thomas Springfield (Ill.) 1960
- GOLDSTIN S. L. and BURGESS J. Spontaneous thrombosis of the internal carotid artery in a 7 year old child. *AMA J. Dis. Child.* 95 (1958) 538
- HALLERVORDEN J. Das Geburtstrauma als Ursache der Entwicklungsheimmung im Kindesalter. *Med. Klin.* (1937) 1224
- Entwicklungsstörungen und frühkindliche Erkrankungen des Zentralnervensystems. In: *Hdb. Inn. Med.* Bd. V/3. J. Springer Berlin Göttingen Heidelberg, 1953
- und MEYER I. E. Cerebrale Kinderlähmung. In: *Hdb. Spez. pathol. Anat. u. Histol.* Bd. 13/4. J. Springer Berlin Göttingen Heidelberg 1956
- HEUBNER O. Über zerebrale Kinderlähmung. *Med. Blatt* 6 (1883) 373
- KUNDRATITZ K., KLALSBERGER E. und ZWENMÜLLER E. Die Bedeutung der Angiographie bei cerebrall gestörten Kindern. *Mischr. Kinderheilk.* 106 (1958) 113
- LANG W. Zur Morphologie der infantilen Hemiplegie. *Dtsch. Z. Nervenheilk.* 185 (1963) 339
- LEFEBVRE J., LEPINTRE J., LAURE C. et PLÉZ J. Résultats de l'angiographie cérébrale au cours des hémiplegies cérébrales infantiles. *Acta radiol.* 46 (1956) 456
- MAIR W. G. Pathological changes in infantile hemiplegia. *Proc. I. Int. Congr. Neuropathol.* Rome 1952
- MEYER I. E. Über Gefäßveränderungen beim fetalen und frühkindlichen Hirnschaden. *Arch. Psychiat. u. Z. Neurol.* 186 (1951) 437
- Zur Ätiologie und Pathogenese des fetalen und frühkindlichen Hirnschadens. *Z. Kinderheilk.* 67 (1949) 123
- Über die Lokalisation frühkindlicher Hirnschaden in arteriellen Grenzgebieten. *Arch. Psychiat. u. Z. Neurol.* 190 (1953) 328

- MYERS D. Carotid thrombosis in childhood. *Arch. Dis. Child.* 35 (1960) 515
- NORMAN R. M. and URICH H. Dissecting aneurysm of the middle cerebral artery as a cause of acute infantile hemiplegia. *J. Path. Bact.* 73 (1957) 580
- PIA H. W. Die Hemisphärektomie in der Behandlung der cerebralen Kinderlähmung. *Dtsch. Z. Nervenheilk.* 181 (1960) 393
- Die operative Behandlung der infantilen Hemiplegie aus prophylaktischer Sicht. *Prophylaxe u. Therapie* Bd 2 S. 1—2. Wien: Braeser 1963
- Neue Wege zur Behandlung der cerebralen Kinderlähmung. *Tgl. Praxis* 1 (1960) 439
- Ätiologie und Pathogenese der infantilen Hemiplegie. *Dtsch. Z. Nervenheilk.* 185 (1963) 357
- PIGAZ J. A. Cerebral angiography in children. *J. Neurosurg.* 9 (1952) 235
- POSER C. M. and TAVERAS J. M. Clinical aspects of cerebral angiography in children. *Pediatrics* 16 (1955) 73
- ROSEGAY H. and WELCH K. Peripheral collateral circulation between cerebral arteries. *J. Neurosurg.* 11 (1954) 563
- SCHWARTZ PH. und FINK L. Morphologie und Entstehung der geburts-traumatischen Blutungen im Gehirn und Schädel der Neugeborenen. *Z. Kinderheilk.* 40 (1925) 427
- STEVENS H. Carotid artery occlusion in childhood. *Pediatrics* 23 (1959) 699
- TAVERAS J. M. and POSER C. M. Roentgenologic aspects of cerebral angiography in children. *Amer. J. Roentgenol.* 111 (1959) 371
- TEBO P. and GOLDENBERG E. D. Thrombosis of internal carotid artery in a 5-year-old child. *A. M. A. J. Dis. Child.* 99 (1960) 228
- TONNIS W. Zit. nach LANG
- VIGOUROUX R., BONDON-RESQUES J., PIGANIOL G. et VIGOUROUX M. Valeur respective de la pneumographie, de l'angiographie et de l'électroencephalographie au cours des encéphalopathies infantiles. *Rev. Neurol.* 90 (1954) 662
- WALTER W. und BRANDT P. Die Angiographie bei den frühkindlichen Hirnschäden. *Acta Neurochir.* 6 (1958) 310
- WIESEL J. Über Arterienveränderungen bei kindlichen Infektionskrankheiten. *Wien. klin. Wschr.* 56 (1961) 15
- WISOFF H. S., YOUNGERS Y. and ROTHBALLER A. Cerebral arterial thrombosis in children. *Arch. Neurol. Psychiat.* 36 (1961) 258
- WOLMAN L. Cerebral dissecting aneurysms. *Brain* 82 (1959) 216
- WILLIE W. G. Acute infantile hemiplegia. *Proc. roy. Soc. Med.* 41 (1948) 459

ARTERIAL ANASTOMOSES OF THE HUMAN BRAIN

A radiographic-anatomic study

by

GERTRAUD WOLLSCHLAEGER and PAUL B WOLLSCHLAEGER

In 1872 COHNHEIM (1) introduced the term 'end arteries' HUBNER (11) injected a colored liquid into the cerebral arteries and observed filling of ligated arteries and very fine collateral vessels He was convinced of the significance of this meningeal network His contemporary, DURET (6), also demonstrated the morphologic presence of these structures with colored gelatin solution BEEVOR (2) defined the areas of distribution of the cerebral arteries He also confirmed the presence of anastomoses between the major arteries The functional significance of these meningeal anastomoses has been well demonstrated by VAN DER ECKEN & ADAMS (18) in 1953 They correlated the clinical symptoms with the findings seen in postmortem injected brains using the anatomic terminology of FORA & LEVI (9) Many authors (5, 8, 12, 13, 14, 15, 16, 17) illustrated the intracerebral collaterals in angiographic studies during life Many of these authors stress the significance in occlusive disease FAY (7) in 1925 had already shown anastomoses in injected brain specimens on stereoröntgenograms He began his study with the original idea of injecting a single artery in order to identify and determine the area of distribution radiographically However, his injection resulted in almost total



Fig 1 a) Base view of postmortem anterior choroidal artery injection. Retrograde filling of posterior cerebral artery (C) via postero-lateral choroidal artery (B). 1 and 2 point to a couple of the numerous anastomoses between the anterior choroidal artery and postero-lateral choroidal artery. b) Base view photograph of left brain hemisphere. The anterior choroidal artery (A) originates from the internal carotid artery (D). It has multiple anastomoses with the postero-lateral choroidal artery (B) especially in the choroid plexus marked 2. The latter artery takes off from the posterior cerebral artery (C).

filling of primarily non injected arteries via the collaterals. He did not show the collaterals in the gross specimen.

Despite the excellent anatomic and radiographic observation of collaterals there is a lack and need for a correlative study to show the anastomoses on angiograms and corresponding anatomic specimens.

The purpose of this paper is to present plainly observed anastomoses or to determine the topographic anatomic site of the anastomoses on the specimen in correlation with the angiogram obtained postmortem.

Material and method. Seventy brains selected at random were studied between October 1961 and June 1962. The patients' age ranged from 23 to 84 years. The area of distribution of the selected artery was carefully examined for its

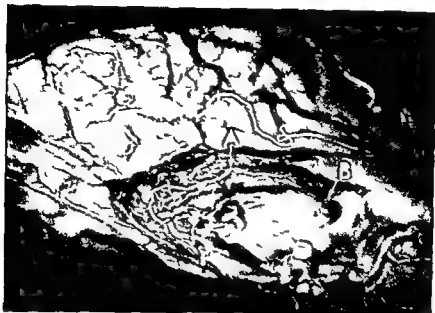


Fig. 2 Lateral view into temporal horn and trigonum and main body of lateral ventricle on the specimen. The postero lateral choroidal artery (A) crosses the trigonum in the direction of the lateral ventricle and approaches the foramen of Monro (B).

topographic relation to the brain surface and the single artery was then injected with a hand pressure of about 100 mm Hg, using a barium gelatin suspension. The injection was immediately terminated when the contrast medium entered the area of distribution of the uninjected artery. Roentgenograms of the fresh unfixed brain were taken in the inferior superior (base view) and lateral positions. A roentgenogram in the r-p projection was avoided because of the instability of the fresh brain in this position. Following angiography of the single injected artery, the brain specimen was usually photographed.

All cerebral arteries were then injected and after 14 days fixation the brain was also roentgenographed and photographed for an unrelated special research project. Evaluation of the cerebral vasculature of 200 brains.

The angiogram of the single injected artery of the fresh brain was subsequently studied and the distribution of this vessel as well as the topographic location of its collaterals were compared with the photographs of the fresh brain. If necessary, further correlations with the photographs and roentgenograms of the totally injected brain were undertaken.

Results and demonstration of anastomoses

Collaterals were observed in 58 specimens. In 12 instances no collaterals could be identified due to the fact that, a) only partial filling of the area of distribution of the single injected artery was achieved because of insufficient in-



Fig 3 Base view photograph of hemilateral specimen preparation. Postero-medial choroidal artery (A) branches early from posterior cerebral artery (B) and courses around brain stem. It enters the third ventricle superiorly and laterally to the pineal gland (C) and continues in an anterior direction on the inferior aspect of the roof of the third ventricle and passes the intraventricular foramen (D) in order to anastomose with the postero-lateral choroidal artery. The postero-lateral choroidal artery (E) originates distal to the postero-medial choroidal artery from the posterior cerebral artery enters the temporal horn and disappears in the trigonum after numerous anastomoses with the anterior choroidal artery (F) in the choroidal plexus.

jection pressure and b) extravasation of the contrast media obscured the collaterals.

In many instances multiple collaterals were noted not only between two different arterial areas of distribution but also from one area to two different areas and within one arterial area itself. In addition it was seen that a single anastomosis may branch and the neighboring anastomosis may also branch. This cross supply was commonly found in the area of the central and post central gyrus. However total elucidation of all these cases is to be the subject of a detailed monograph. The complexity of the arterial anastomoses will be demonstrated in 5 different cases.

In most instances no attempt was made to dissect or elevate the anastomotic branches from the depth of the sulci to the visible surface of the brain, because this would have produced an unreliable picture due to the unfolding of the vessels which would not correspond well to the topographic location of the anastomoses when compared with the postmortem angiogram of the fresh brain.

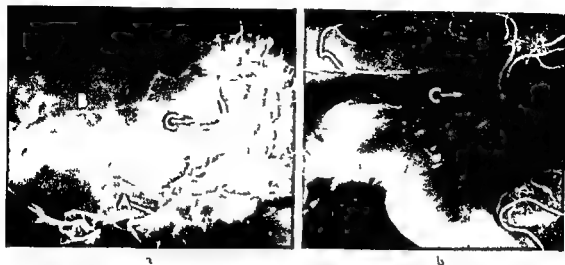


Fig. 4 a) Lateral view of postmortem roentgenograms. The posterior cerebral artery (A) is injected and joins the anterior cerebral artery (B) via the callosal branch (C). b) Radiograph of sagittal brain section (mid line cut). The callosal branch (C) connects the posterior cerebral artery with the anterior cerebral artery.

Numerous injections of the anterior cerebral artery and middle cerebral artery were performed and their anastomoses studied precisely and compared with the work of VAN DER ELKEN & ADAMS 18, and MOUNT & TAVIRAS (14). A description of these anastomoses will therefore be omitted.

We were particularly interested in anterior choroidal artery injections. This vessel originated from the internal carotid artery in most cases. Its outer diameter at its origin measured 0.5 mm which corresponds well with the average outer diameter of the anterior choroidal artery registered in a series of 376 measurements by us (19). In this series the maximum diameter of the anterior choroidal artery reached 0.9 mm in a case of interventricular meningioma fed by the anterior choroidal artery. These data are identical with the measurements by CARPENTER *et coll.* (3), who also described the anatomy and the anastomoses. According to this author, and to ABBIE (1), anastomoses occurred between the anterior choroidal artery and the posterolateral choroidal artery, either rostral to the lateral geniculate body or on the surface of the aforementioned structure or at both sites, in 36 of 15 instances. The anastomoses which were seen on the roentgenograms and dissected specimen of this study were similar to those reported by CARPENTER *et coll.* (3).

There are immediate anastomoses between the anterior choroidal artery and the posterolateral choroidal artery in the choroid plexus of the lateral portion of the trigonum. In the same region there are multiple small anastomoses. This vessel then takes its course out of the trigonum into the main body of the lateral ventricle and heads towards the foramen of Monro, where it



Fig 5 View into the interhemispheric fissure from posterior. The callosal branch (arrow) curves over the splenium of the corpus callosum and links the anterior and posterior cerebral arteries.



Fig 6 Postmortem roentgenogram lateral view. The superior cerebellar artery (A) is injected and anastomoses with the inferior posterior cerebellar artery (B), at 1 and 2. Note relationship of the inferior posterior cerebellar artery (B) to the roof of the fourth ventricle.

meets with the postero medial choroidal artery. The latter originates separately either from the proximal posterior cerebral artery or the distal basilar artery. From there its pathway can be observed around the brain stem approaching the roof of the third ventricle superiorly and slightly laterally to the pineal gland. Further on it enters the foramen of Monro with very fine branches which anastomose with the postero lateral choroidal artery in the lateral ventricle. This would indicate that all choroidal arteries should be considered as a functional unit.

During the injection of posterior cerebral arteries we noticed filling of a fine callosal branch of the posterior cerebral artery which combines with the pericallosal artery. This anastomosis is anatomically located in the midline while the vessel curves over the splenium of the corpus callosum. Another interesting connection occurs between the parietal branch of the posterior cerebral artery and the cuneal branch of the anterior cerebral artery. The cuneal branch of the anterior cerebral artery on the other hand joins the ascending parietal branch of the middle cerebral artery and produces retrograde filling of the middle cerebral artery distribution. The topographic anatomic location of this anastomosis is recognized on the precuneus and cuneus, at the medial surface of the hemisphere.



Fig. 7 a) Basal view photograph of left cerebellum after total injection. The anastomoses are seen at the great horizontal fissure at 1 and 2. b) Lateral view of cerebellum. The lateral branch of the superior cerebellar artery (A) anastomoses with the inferior posterior cerebellar artery at 1.

We also achieved anastomosis between the posterior temporal branch of the posterior cerebral artery and the posterior temporal branch of the middle cerebral artery. This anastomosis is located in the parieto-occipital sulcus.

Single injection into cerebellar arteries likewise produced very informative anastomoses, a few of which are illustrated.

Branches of the posterior cerebellar artery frequently cause filling of twigs of the anterior inferior cerebellar or superior cerebellar arteries. The inferior posterior cerebellar artery winds around the medulla oblongata in a posterior medial direction, so that a loop is formed with the convexity in the apex of the rhomboidal fossa. This loop has a close relation to the roof of the fourth ventricle. In this region, anastomoses with the inferior posterior cerebellar artery of the opposite side are commonly observed. The vessel transcends in a posterior inferior direction parallel to the anterior inferior surface of the cerebellum, and crosses over the medial portion of the tonsil. On the inferior lateral border of the tonsil, the vessel divides into a medial and lateral branch. The lateral branch passes over the biventer and its multiple branches cross the great horizontal fissure to the superior surface of the cerebellum to anastomose with the lateral branches of the superior cerebellar artery. The medial branch of the inferior posterior cerebellar artery often combines with the medial branch of the superior cerebellar artery on the medial portion of the posterior superior lobule.

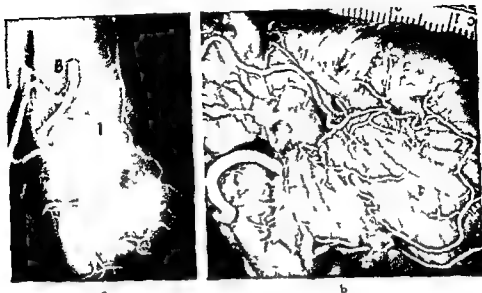


Fig 8 a) Postmortem roentgenogram base view of cerebellum. The inferior posterior cerebellar artery (B) is injected and shows anastomoses with the superior cerebellar artery (A) at 1 and 2. b) Photograph of brain specimen in (a). The anastomoses 1 and 2 are visible with it being elevated from the fissure.

Discussion

The postmortem injection of barium gelatin suspension was used by many authors in the past to demonstrate arterial anastomoses. Usually, however, the anastomotic channels were analyzed only on anatomic specimens or only on roentgenograms of the specimens. Of equal importance are photographs of the surface of the injected brain to verify the topographic region of the radiographically recognized anastomoses. Surface photographs make the dissection and elevation of the anastomoses and tributaries unnecessary in most instances. Any dissection and elevation of vessels from the depths of the sulci will result in an unfolding which will demonstrate the total length of the vessel, but this will exceed by 2 to 4 times the length of the previously occupied brain surface. Any artificial placement of the unfolded vessels will produce an unrealistic impression. However, in some instances a dissection is certainly necessary. The use of the freshly injected specimen for radiographic study has the great advantage that the same brain can be used for a subsequent study, if the final total injection is done prior to coagulation of the barium gelatin suspension. However, a slight disadvantage of using fresh brains is the instability; this was overcome by wrapping the brain in gauze and by selecting

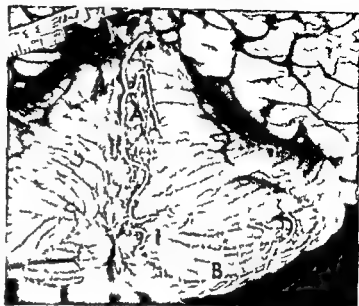


Fig. 9 Brain specimen rostral view of cerebellum. The dorso-medial branch of the superior cerebellar artery (A) anastomoses at the great horizontal fissure (1) with the posterior inferior cerebellar artery (B).

the lateral and infero superior position (base view) for the roentgenography. The injection of a single artery may not always result in filling of all possible anastomoses from the injected area of distribution to another area. When this happens it is necessary to repeat the injection of the same artery in a different brain.

Collaterals were seen on the roentgenograms of all 58 brains. A very striking finding was the frequent presence of double anastomotic channels in the prerolandic, rolandic and postrolandic regions. An evaluation of this observation and the anatomic variation of the tributaries which feed the arterial anastomoses in human brain over this region will be included in the monograph.

Our special interest was directed to the anterior and posterior choroidal arteries and their anastomoses. Most authors agree that the anastomoses between the postero lateral choroidal artery and the anterior choroidal artery are located in the temporal horn and in the choroid plexus of the trigonum extending into the posterior portion of the body of the lateral ventricle. They also agree that the postero medial choroidal artery arises from the posterior cerebral artery or basilar artery proximal to the origin of the postero lateral choroidal artery. Through dissection of 9 specimens for demonstration of the topographic site of the anastomoses, it was noted in most instances that the postero medial choroidal artery originates superiorly or laterally superiorly from the posterior cerebral artery and then turns medial to the posterior cerebral artery, trails around the brain stem, and enters laterally and superiorly

to the pineal body and laterally and inferiorly to the internal cerebral vein of the third ventricle. Usually, it was easy to follow this artery to the foramen of Monro. According to GALLOWAY & GREITZ (10) there is no connection between the anterior choroidal artery and the postero medial choroidal artery. However, we observed and demonstrated its filling from the anterior choroidal artery by way of the postero lateral choroidal artery. We agree with these authors that the postero lateral choroidal artery does not come to the sagittal midline of the brain, as does the postero medial choroidal artery. The clinical significance of the anastomoses between the anterior choroidal and posterior choroidal arteries is important in the presence of tumors of the choroid plexus, intraventricular meningiomas, and in the presence of A V malformations. The anastomoses from the posterior cerebral artery to the middle cerebral artery and to the anterior cerebral artery are well known. Variations of the topographic site were observed. The anastomoses on the cerebellum have been well described by KRAYENBUHL & YASARGIL (12). Concerning the anastomoses over the cerebellum, in one instance we noticed a connection between the left inferior posterior cerebellar artery and the left superior cerebellar artery and the right posterior cerebral artery. Such anastomoses complicate the localization of A V malformations in these areas of distribution. The space available here does not allow a complete description of the enormous variety of anastomoses on the cerebellum.

Acknowledgements

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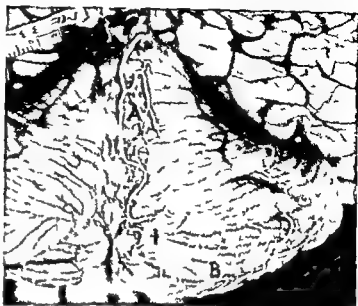
SUMMARY

Arterial anastomoses of the human brain were demonstrated on post mortem angiograms with correlation of the topographic anatomical region on brain specimens. Special attention was directed to the anterior choroidal artery and its anastomoses with the posterior choroidal arteries.

ZUSAMMENFASSUNG

Arterielle Anastomosen des menschlichen Gehirns wurden mittels postmortaler Angiographie dargestellt und gleichzeitig auf die topographisch anatomische Lagebeziehung am Gehirn hingewiesen. Dabei wurde der Arteria chorioidealis anterior nebst ihrer Anastomosen mit der Arteria chorioidealis posterior besondere Beachtung gezollt.

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RÉSUMÉ

Les anastomoses artérielles du cerveau humain ont été étudiées par angiographie post mortem confrontée avec l'étude topographique de pièces d'autopsie. L'auteur a étudié spécialement les anastomoses de l'artère choroidienne antérieure avec les artères choroidiennes postérieures.

REFERENCES

1. AUDIE A. A. The blood supply of the lateral geniculate body with a note on the morphology of the choroidal arteries. *J Anat* 67 (1933) 491.
2. BLYVOR C. I. The cerebral arterial supply. *Brain* 30 (1907) 403.
3. CARLISLER M. B., NOBACH C. R. and MOSS M. L. The anterior choroidal artery. *Arch Neurol Psychiat* 71 (1954), 714.
4. COHNHEIM J. Untersuchungen über die embolischen Prozesse. August Hirschwald Berlin 1872. Cited by IONNIS and SCHIEFER.
5. DICKER K. und HOLZLER L. Gefäßverschlüsse im Carotis und Vertebrobasiliogrammen. *Fortchr Röntgenstr* 80 (1954) 565.
6. DURET H. Recherches anatomiques sur la circulation de l'encéphale. *Arch physiol norm et path*. Second series 1 (1874) 60-316.
7. EAY T. The cerebral vasculature. Preliminary report of study by means of roentgen ray. *J Amer med Ass* 84 (1925) 1727.
8. EIRING E. H. and SUSSMAN B. J. Spontaneous occlusion of the middle cerebral artery. *Neurology* 6 (1956) 529.
9. FOIX C. and LÉVY M. Ramollissement Sylvius. *Rev neurol* 21 (1927) 51.
10. GALLOWAY J. R. and GRILITZ T. The medial and lateral choroid arteries. An anatomic and roentgenographic study. *Acta radiol* 53 (1960) 353.
11. HUBNER O. Zur Topographie der Ernährungsgebiete der einzelnen Hirnarterien. *Zbl med Wiss* 10 (1872) 817.
12. KRAVENBUHL H. und YASARGIL M. G. Die vasculären Erkrankungen im Gebiete der Arteria vertebralis und Arteria basialis. Georg Thieme Verlag Stuttgart 1957.
13. MOUNT L. A. and TAYLOR J. M. A study of the collateral circulation of the brain following ligation of the internal carotid artery. *Trans Amer neurol Ass* 78 (1953) 47.
14. — — Arteriographic demonstration of the collateral circulation of the cerebral hemispheres. *AMA Arch Neurol Psychiat* 78 (1957) 235.
15. ROSEGAY H. and WELCH K. Peripheral collateral circulation between cerebral arteries. A demonstration by angiography of the meningeal arterial anastomoses. *J Neurosurg* 11 (1954) 363.
16. LATELMAN M. Pathways of cerebral collateral circulation. *Radiology* 75 (1960) 349.
17. IONNIS W. und SCHIEFER W. Zirkulationsstörungen des Gehirns im Serienangiogramm. pgs 46 und 139. Springer Verlag Berlin 1959.
18. VAN DER ELKEN M. and ADAMS R. D. The anatomy and functional significance of the meningeal arterial anastomoses of the human brain. *J Neuropath exp Neurol* 12 (1953) 132.
19. WOLLSCHLAEGER G. and WOLLSCHLAEGER P. B. The anterior choroidal artery. To be published. Presented at the meeting of the Neuroradiological Society of North America, Atlantic City, U.S.A., June 1965.

ANTERIOR CEREBRAL/INTERNAL CAROTID ARTERY AND MIDDLE CEREBRAL/INTERNAL CAROTID ARTERY RATIOS

by

PALL B WOLLSCHLAEGER and GERTRAUD WOLLSCHLAEGER

During the interpretation of carotid angiograms the problem of whether one is dealing with hypoplastic spastic or arteriosclerotic arteries often arises and it is difficult to define what should be considered an average diameter for a vessel of the circle of Willis as stated by HARGIS et coll. With respect to the first category the pathologist generally defines hypoplasia as a congenital defective formation in which there is incomplete development of an organ or a part. ALPERS and his co authors remarked that stringlike vessels with a diameter of 1 mm are hypoplastic but functional.

The evaluation of an artery from the standpoint of the diameter of the lumen may be done by measuring the involved vessel. PALLIE & SAMARASINGHE have contributed interesting and valuable information from studies of 12 brain specimens. On the other hand one might prefer correlations of branching arteries like TURNER who reported the results of arterial radius measurements of the vertebral arteries and basilar artery.

The purpose of this paper is to arrive at measurements of single arteries

RÉSUMÉ

Les anastomoses artérielles du cerveau humain ont été étudiées par angiographie post mortem confrontée avec l'étude topographique de pièces d'autopsie. L'auteur a étudié spécialement les anastomoses de l'artère choroidienne antérieure avec les artères choroidiennes postérieures.

REFERENCES

1. ABUL A. A. The blood supply of the lateral geniculate body with a note on the morphology of the choroidal arteries. *J. Anat.* 67 (1933) 491.
2. BELVOR C. L. The cerebral arterial supply. *Brain* 30 (1907), 103.
3. CANNISTER M. B., NOBACK C. R. and MOSS M. L. The anterior choroidal artery. *Arch. Neurol. Psychiat.* 71 (1954) 714.
4. COHNHEIM J. Untersuchungen über die embolischen Prozesse. August Hirschwald, Berlin 1872. Cited by TONNIS and SCHIEFER.
5. DECKER K. und HOLZLER E. Gefäßverschlüsse im Carotis- und Vertebraisangiogramm. *Dtschr. Röntgenstr.* 80 (1954) 565.
6. DURET H. Recherches anatomiques sur la circulation de l'encéphale. *Arch. physiol. norm. et path.* Second series 1 (1874) 60-316.
7. ILL T. The cerebral vasculature. Preliminary report of study by means of roentgen ray. *J. Amer. med. Ass.* 84 (1925) 1727.
8. LIRING E. H. and SUSSMAN B. J. Spontaneous occlusion of the middle cerebral artery. *Neurology* 6 (1956) 529.
9. LOIS C. and LEVY M. Ramollissement Sylius. *Rev. neurol.* 21 (1927) 51.
10. GALLOWAY J. R. and CREITZ T. The medial and lateral choroid arteries. An anatomic and roentgenographic study. *Acta radiol.* 53 (1960) 353.
11. HEUBNER O. Zur Topographie der Ernährungsgebiete der einzelnen Hirnarterien. *Zbl. med. Wiss.* 10 (1872) 817.
12. KRAYENBUHL H. und VASARGIL M. G. Die vasculären Erkrankungen im Gebiete der Arteria vertebralis und Arteria basialis. Georg Thieme Verlag, Stuttgart 1957.
13. MOUNT L. A. and LAVIRAS J. M. A study of the collateral circulation of the brain following ligation of the internal carotid artery. *Trans. Amer. neurol. Ass.* 78 (1953) 47.
14. — — Arteriographic demonstration of the collateral circulation of the cerebral hemispheres. *A.M.A. Arch. Neurol. Psychiat.* 78 (1957) 235.
15. ROSE GAY H. and WELCH K. Peripheral collateral circulation between cerebral arteries. A demonstration by angiography of the meningeal arterial anastomoses. *J. Neurosurg.* 11 (1954) 363.
16. TATELMAN M. Pathways of cerebral collateral circulation. *Radiology* 75 (1960) 349.
17. TONNIS W. und SCHIEFLER W. Zirkulationsstörungen des Gehirns im Serienangiogramm. pgs 46 und 139. Springer Verlag, Berlin 1959.
18. VAN DER LECHE M. and ADAMS R. D. The anatomy and functional significance of the meningeal arterial anastomoses of the human brain. *J. Neuropath. exp. Neurol.* 12 (1953) 132.
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by

PAUL H WOLLSCHLAEGER and GERTRAUD WOLLSCHLAEGER

During the interpretation of carotid angiograms the problem of whether one is dealing with hypoplastic spastic or arteriosclerotic arteries often arises and it is difficult to define what should be considered an average diameter for a vessel of the circle of Willis as stated by KIRGIS et coll. With respect to the first category the pathologist generally defines hypoplasia as a congenital defective formation in which there is incomplete development of an organ or a part. ALPFERS and his co authors remarked that stringlike vessels with a diameter of 1 mm are hypoplastic but functional.

The evaluation of an artery from the standpoint of the diameter of the lumen may be done by measuring the involved vessel. PALLIE & SAMARASINGHE have contributed interesting and valuable information from studies of 12 brain specimens. On the other hand one might prefer correlations of branching arteries like TURNER who reported the results of arterial radius measurements of the vertebral arteries and basilar artery.

The purpose of this paper is to arrive at measurements of single arteries

SCHEME OF MEASUREMENTS

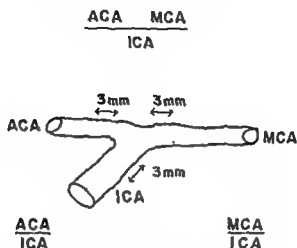


Fig. 1 Diagrammatic representation of method of measurement

and also to establish ratios between the branching vessels for evaluation of the presence of a hypoplastic artery.

A total of 1 050 measurements of the outside diameter of the anterior cerebral artery (ACA), middle cerebral artery (MCA), and internal carotid artery (ICA) were made on 175 unselected brain specimens, which had been injected with a barium gelatin suspension immediately after removal. After the days of fixation the diameters were read at points 3 mm proximal and 3 mm distal to the division of the internal carotid artery into the anterior cerebral and middle cerebral arteries (Figs 1 and 2). Similar measurements were made in the arterial phase of the half axial x-ray projection of 124 angiograms.

The results were as follows. In the angiographic studies we were measuring the intraluminal diameters, which amounted in average (130 angiograms) to 2.03 mm for ACA, 2.87 mm for MCA and 3.88 mm for ICA, as compared with the postmortem diameters (352), which were 1.7 mm for ACA, 2.5 mm for MCA and 3.7 mm for ICA. The differences of angiographic versus postmortem data were 0.33 mm for ACA (16%), 0.37 mm for MCA (13%) and 0.18 mm for ICA (±6%).

From the measurements listed previously, the average ratio was obtained by calculation on the postmortem and *in vivo* studies as follows:

$$\begin{array}{l} \text{ACA/ICA postmortem } 0.459, \text{ in vivo } 0.5241, \text{ difference } 0.0651 \\ \text{MCA/ICA postmortem } 0.675, \text{ in vivo } 0.7395, \text{ difference } 0.0645 \\ \frac{\text{ACA} + \text{MCA}}{\text{ICA}} \text{ postmortem } 1.135, \text{ in vivo } 1.2637, \text{ difference } 0.1287 \end{array}$$



Fig 2 The normal trifurcation. Sections of base view of postmortem angiogram (top) of base view of total brain specimen after injection (centre) and of circle of Willis of same specimen (bottom). Site of measurements indicated by black lines on bottom section. A = first portion of anterior cerebral artery. M = first part of middle cerebral artery. ICA = internal carotid artery.

The 10–20% value increase of the *in vivo* measurements can be explained by roentgenographic magnification and also by the fact that we were dealing with two different populations. The single postmortem figures for ACA were compared with the macroscopic brain description of the neuropathologic service respective to hypoplasia of this vessel. There we learned that anterior cerebral arteries with diameters of about one millimeter or less have been called hypoplastic. This corresponds with the figures mentioned in the literature. From comparison of the ACA diameter in our angiographic series with independent reports of the neuropathologic department on the same diseased patients we concluded that an angiographic ACA diameter of 1.2 mm or less may be considered hypoplastic. Therefore our postmortem single diameter value concerning hypoplasia of the anterior cerebral artery can be applied in angiography.



Fig. 3 a) Right carotid angiogram with compression of contralateral internal carotid artery half axial a p view. Filling of right and left anterior cerebral arteries. The right is hypoplastic (arrow) b) Base view of brain specimen from same patient. Right anterior cerebral artery is hypoplastic (arrow)

The average postmortem ACA/ICA ratio was 0.45, but decreased to 0.28, when there was evidence of hypoplasia, in 31 instances. In 10 angiographic cases with a hypoplastic anterior cerebral artery the ACA/ICA ratio showed only minimal variations and was about 0.29. This proves, likewise, that our postmortem ratio values can be used in *in vivo* evaluation of arterial hypoplasia.

We are aware of the risk that figures arrived at from postmortem material may not be entirely reliable. We have studied a patient at *in vivo* angiography and at necropsy (Fig. 3). Both *in vivo* and postmortem ACA diameters measured 1 mm. The *in vivo* ACA/ICA ratio was 0.28 and the postmortem ACA/ICA ratio 0.25. The individual measurements as well as the ratios were just about equivalent.

In cases of arteriosclerosis, our figures vary markedly from one brain specimen to another (Fig. 4). It is known that the diameter alters within short distances and that the measurements at a given point may meet normal arterial width. For this reason single data and ratios would fall within normal ranges. It might also be necessary to consider the factor that *in vivo* and postmortem data in arteriosclerosis represent lower and upper range extremes for one particular case, which is explained by the increasing wall thickness due to irregular atheromatous plaques.



Fig 4 a) Angiogram taken with half axial a-p projection. Marked beading of intracranial arteries. This appearance is identical with severe arteriosclerotic changes. b) Base view of brain specimens from same patient. Diameters of vessels variable due to atheromatous plaques (arrow)

The values obtained for single diameters and ratios could not be employed in cases of arterial spasm. One reason is the limited number of cases with true and proven occurrence of spasm. ECKER & RIEMENSCHNEIDER have discussed the reliability of angiographic evidence of spasm in the internal carotid arteries and their main branches and have listed an interesting grading of arterial spasm. They also emphasized the fact that it is necessary to see a vessel in two projections at different series and times. Other authors (DECKER, HERMANN, HUBER, KRAEVENBUHL) were able to verify the presence of spasm so called post-traumatic arterial diameter fluctuations by follow up angiography of their patients. However in many cases it is impossible to re-examine patients and therefore other means of definition must be found. We shall certainly pursue this field further. Second it is quite possible in this category that one measured diameter and its calculated ratio at a given point of the branching artery is not sufficient since spasm frequently exceeds one segment or more of an artery and often involves two branching vessels. Additional measurements on specified distances may be necessary and a subtraction factor perhaps worked out in order to differentiate arterial spasm from hypoplastic vessels.

Acknowledgement

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SUMMARY

Average diameters of the anterior cerebral artery, middle cerebral artery and internal carotid artery close to the bifurcation of the internal carotid artery and their ratios were established, and an attempt was made to apply the obtained data, especially in cases of hypoplasia, and also though to a limited extent, in cases of spasm and arteriosclerosis. The ratios arrived at post mortem can be applied practically in angiographic studies in vivo.

ZUSAMMENFASSUNG

Die Durchmesser der Arterien cerebri anterior, media und carotis interna dicht bei der Verzweigung der Arteria carotis interna wurden notiert und deren Verhältnisswerte aufgestellt. In einer Versuchsreihe wurden die erarbeiteten Werte in Fällen von arterieller Hypoplasie und im begrenzten Umfang in Fällen mit Gefäßspasmus und Arteriosklerose angewandt. Die an Gehirnpräparaten erhaltenen Verhältnisswerte können auf die zerebrale Angiographie in vivo übertragen werden.

RÉSUMÉ

L'auteur a mesuré le diamètre moyen des artères cérébrale antérieure, cérébrale moyenne et carotide interne et il a calculé leur rapport et a essayé d'appliquer ces résultats aux cas d'hypoplasie et aussi dans une certaine mesure à des cas de spasme et d'artériosclérose. On peut pratiquement appliquer aux études angiographiques in vivo les rapports établis post mortem.

REFERENCES

- ALPERS B. J., BERRY R. G. and PADDISON R. M.: Anatomical studies of the circle of Willis in normal brain. *A. M. A. Arch. Neurol. Psychiat.* 81 (1959) 409.
- DECKER H.: Der Spasmus der Arteria carotis interna. *Acta radiol.* 46 (1956) 351.
- ECKER A. and RIEMENSCHNEIDER P. A.: Arteriographic evidence of spasm in cerebral vascular disorder. *Neurology* 3 (1953) 495.
- HERMANN E.: Die Deutung der Einengung des Supraclinoidalen Syphonabschnittes der Arteria Carotis Interna im Arteriogramm. *Dtsch. Z. Nervenheilk.* 183 (1961) 99.
- HÜBER P.: Posttraumatische Kaliberschwankungen der Hirngefäße im Angiogramm. *Fortschr. Röntgenstr.* 98 (1963) 292.
- KIRGIS H. D., LLEWELLYN R. C. and PEEBLES E. M.: Functional trifurcation of the internal carotid artery and its potential clinical significance. *J. Neurosurg.* 17 (1960) 1062.
- KRAYENBUHL H.: Beitrag zur Frage des Cerebralen Angiographischen Insultes. *Schweiz. med. Wschr.* 90 (1960) 961.
- PALLIE W. and SANARASINIE D. D.: A study in the quantification of the circle of Willis. *Brain* 85 (1962) 569.
- TURNER R.: A comparison of theoretical measurements with observed angles between the vertebral arteries and their function to form the basilar artery. *Anat. Record* 129 (1957) 243.
- ZÜLCH K. J.: Gibt es Spasm der Hirngefäße? *Medizinische* 14 (1959) 622.

CEREBRAL EMBOLI COMPLICATING CEREBRAL ANGIOGRAPHY

by

LESLIE M ZATZ and ANTHONY M IANONE

A wide variety of complications has been reported following cerebral angiography, ranging from minimal neurologic deficits to hemiplegia, convulsions and death. In the past such untoward sequelae have usually been attributed to the hyperosmotic iodinated contrast agents employed. In recent years the contrast agents originally employed have been discarded and replaced by agents with a considerably larger margin of safety. Any evidence that known complications in man are produced by factors other than the contrast agents and are preventable would be of importance in decreasing the risk of this procedure. This paper presents evidence that emboli may be the responsible agent in certain complications; that embolic accidents may be directly related to technical factors; and that preventive measures may decrease the incidence of such complications.

Diagnostic criteria

The recognition of an embolic complication depends on the identification of an intracerebral occlusion which occurred during the procedure. An intracerebral arterial occlusion is identified by (1) the absence of vessels in an area

usually supplied by a particular vessel, (2) the presence of an abrupt termination of a vessel prior to its normal arborization, or (3) the retrograde flow of contrast material into an occluded area from leptomeningeal collaterals (10, 18, 19). While an occlusion of the middle cerebral artery may be easily recognized by its lack of filling, an absence of filling of one of its branches may not be observed because of overlap of other branches. In such an instance, criteria (2) and (3) are the most important. The filling of the occluded vessel and of the collateral vessels is slower than that of normal arteries. These vessels may not be seen until the small vessel or venous phase of the study.

To demonstrate small vessel occlusions, serial angiography covering seven or eight seconds, at least in the lateral projection, is a necessity. This usually consists of two films per second for three seconds followed by one film per second for an additional four seconds. Stereoscopic lateral films are obtained by alternate exposure with one of a pair of roentgen tubes. This has been of great help in separating superimposed vessels and in tracing collateral vessels into an area of occlusion.

The occlusion can be deduced to be embolic under certain circumstances. The most reliable evidence is the presence on one film of a vessel which is then found to be occluded on a second film a few minutes later. In other cases, the actual process of occlusion may be demonstrated on successive films in the same angiographic series. Failure to demonstrate a vessel at one examination during which a neurologic complication developed is also suggestive if the vessel was present on films from a previous angiography during the same illness. The finding of multiple small vessel occlusions in different vessels at the onset of a complication most likely indicates multiple emboli. When a repeat angiography shows the site of a previously identified obstruction to be smooth and regular after lysis of the occlusion, the original lesion was more likely embolic than thrombotic.

Case reports

Case 1. A 49-year-old right-handed female with a history of a previously resected bronchiolar carcinoma was examined because of increased intracranial pressure and major motor seizures. The ultimate diagnosis proved to be carcinomatous infiltration of the meninges. During her evaluation percutaneous left carotid angiography was done. A halfaxial series was exposed first followed by a lateral series. Immediately upon completion of the examination the patient was noted to be dysphasic and to have a right hemiparesis. The arteriogram revealed that an occlusion of a branch of the middle cerebral artery to the region of the motor cortex had occurred between the first and second examinations probably due to an embolus (Fig. 1). Within 3 hours the neurologic defects had improved considerably and by 24 hours had completely disappeared.

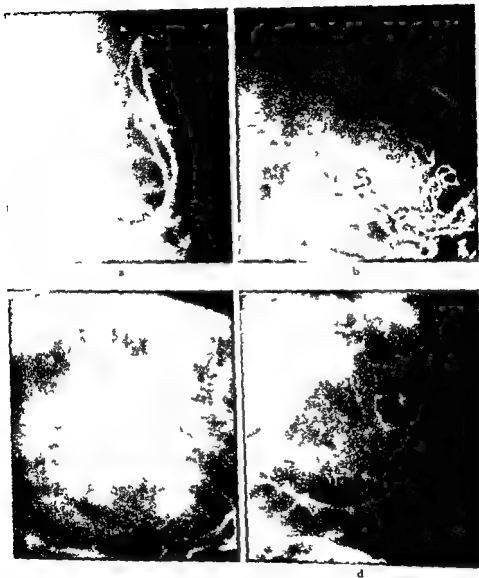


Fig 1 Case 1 a) Arterial phase in half axial projection from first injection. Six branches of middle cerebral artery run transversely in the Sylvian fissure. No evidence of collateral vessels in subsequent films of the series. b) c) and d) Selected early, mid and late arterial films from lateral series following the half axial study. In (b) no middle cerebral branches in frontoparietal area. A small slip vessel may be identified by its characteristically convoluted course. Only four branches of middle cerebral artery emerge from Sylvian fissure. In contrast to the six branches noted in the first series. The indicated vascular occlusion between the first and second series. In (c) several arterial branches begin to fill the retrograde fashion into the avascular area and in (d) progress further toward Sylvian fissure. The retrograde filling of the occluded branches indicates collateral anastomoses.

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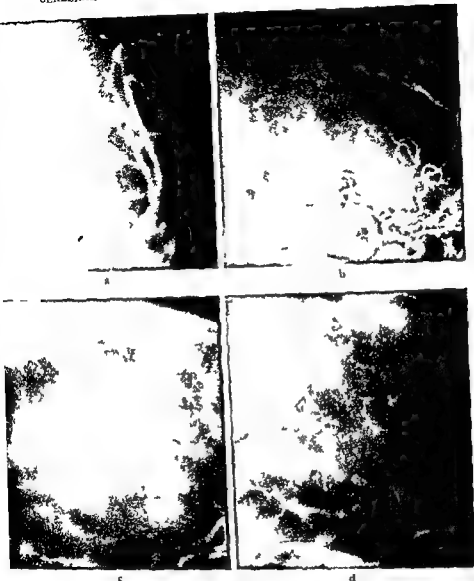


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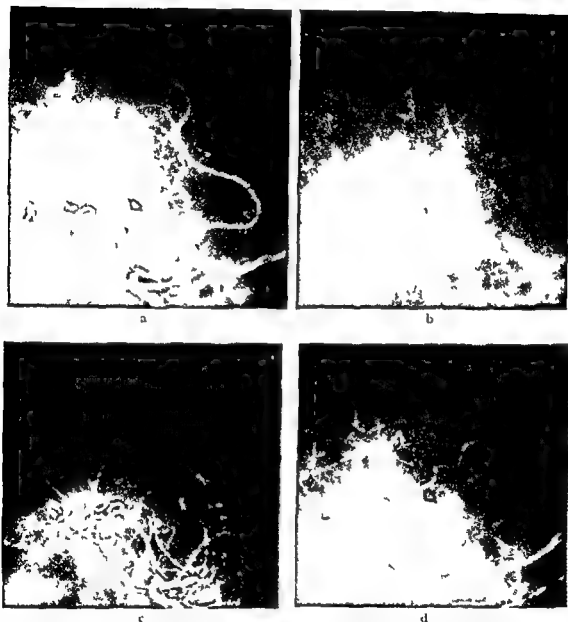


Fig. 2 Case 2. (a) and (b) Early arterial and early venous phases from first lateral series. Complete filling of middle cerebral branches in (a) arrow indicates the point subsequently shown (c) to be occluded. In (b) persistence of candelabra shaped branches of this vessel in the venous phase indicates stasis resulting from the occlusion which apparently occurred at injection. (c) and (d) Early and late arterial phases from a second lateral series a few minutes later. Following this the patient's clinical signs of a neurologic complication were first noted. In (c) only insular portion of occluded vessel can be identified. The peripheral branches are not filled producing an avascular region in the frontoparietal area. In (d) the occluded vessel is filled to point indicated by open arrow. Subsequent films showed no progression of contrast material beyond this point suggesting that this was the site of the occlusion. The solid arrow points to a peripheral branch of the occluded vessel which filled in retrograde fashion from anastomoses with the anterior cerebral artery. This same branch can be seen in frames (a) and (b) filling in normal progade fashion. This illustrates the immediate availability of leptomeningeal collateral anastomoses in the case of an occlusion.

Case 2 A 60 year-old right handed male with a previous diagnosis of a hypernephroma was examined because of a focal right sided weakness one week prior to admission. Physical examination showed a slight right upper monoparesis with decreased proprioceptive and vibratory sensation in the right hand. A percutaneous left carotid arteriography was done. Because of what was thought to be a technical error 2 lateral series were exposed in rapid succession. Immediately afterwards the patient was noted to be aphasic with a right central facial paresis and a complete right upper monoplegia. The films showed that an occlusion of a frontoparietal branch of the middle cerebral artery in the region of the motor cortex had occurred at the time of the first injection (Fig 2). This was presumed to be embolic. Within 3 days after the angiography the patient had regained considerable strength in the extremity. The aphasia and a minimal paresis in the right arm were however still present 14 days later.

The first series of lateral films demonstrated the occlusion of the vessel after some contrast material had entered it (Fig 2 a and b). This produced sudden stasis in the vessel and the branches remained filled with contrast medium for a prolonged period of time. The second lateral series of films then showed the vessel to be totally occluded (Fig 2 c and d). The peripheral branches which had filled in normal fashion minutes before were now seen to fill in a retrograde direction via collateral vessels from the anterior cerebral artery.

Comments The carotid angiographies in Cases 1 and 2 were performed by percutaneous puncture of the common carotid artery with a disposable 18 gauge $3\frac{1}{2}$ inch spinal needle. Following puncture of the vessel and between injections the stylet was replaced in the cannula. While this technic was in use a total of 8 cases in a series of 150 consecutive cerebral angiographies were seen in which small peripheral branch occlusions were identified following the procedure. Of these occlusions 5 were asymptomatic. Most of them were found in patients who were examined for conditions other than ischemic cerebrovascular disease and two were in patients in their second decade.

No source of emboli was apparent in the neck vessels nor was there evidence of a subintimal injection at the site of the needle. Speculation as to the source of the emboli therefore led us to examine the needle used. The needle has a dead space in the hub around the stylet in which clots may form. Fragments of clot may be extruded from the cannula by insertion of the stylet or by the forceful injection of contrast material. The incidence of this complication decreased markedly when instead of replacing the stylet a transparent connecting tube was attached to the cannula following puncture of the vessel. The tubing and the needle were flushed frequently with sterile saline.

Both cases demonstrated the immediate availability of collateral channels in the leptomeninges between the anterior cerebral and middle cerebral arteries. Within minutes of the occlusion the collateral vessels were of sufficient calibre to fill the peripheral branches of the occluded vessel with contrast material.

Case 3 A 60 year old right handed female was investigated because of a 6 months history of episodic vertigo and ataxia precipitated by lateral rotation of the head. Except for a blood pressure of 160/110 and the residual of an old left facial palsy her examination was normal. Aortography was done to demonstrate the extracranial cerebral vessels. A preshaped yellow Ödman catheter with several side holes was introduced into the right femoral artery without difficulty and passed into the ascending aorta. Three injections of Renografin 76° (Methyl glucamine diatrizoate) were made for a p and oblique projections using a pressure injector. The tip of the catheter was then placed selectively in the right carotid artery and 2 injections of Renografin 60° were made by hand for lateral and a p views of the intracranial vessels. After further manipulation during which it was necessary to insert the guide wire the catheter

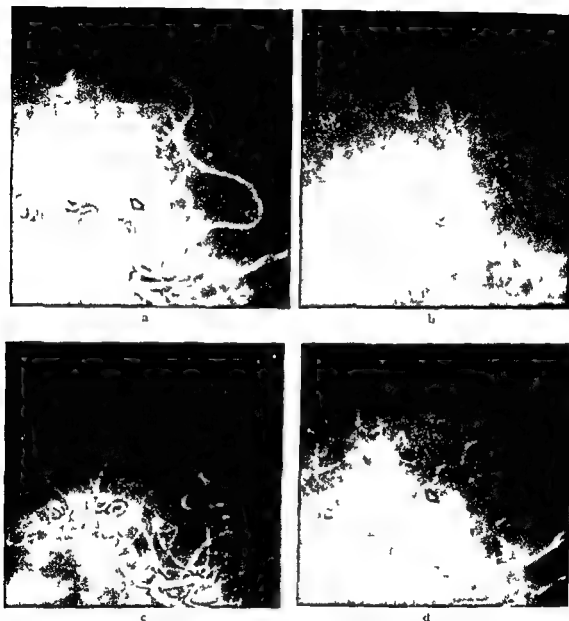


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proved slightly and she began to respond to verbal commands. At the time of discharge 13 days after the angiography the motor aphasia was present without evidence of the right hemiparesis.

Comments. In Case 3 the films revealed occlusions in branches of the middle and anterior cerebral arteries in a patient who developed signs of focal left hemisphere dysfunction following an injection into the left common carotid artery. The indispensability of serial films was demonstrated by this case. In the early lateral films branches of the middle cerebral artery on the opposite side which had filled via the anterior communicating artery appeared to fill the area shown on later films to be avascular. The occlusions in the anterior cerebral artery were obscured by the opposite anterior cerebral artery and it was not until the delayed films that the occlusion became apparent.

The finding of multiple small occlusions suggested that the injection had produced multiple emboli. The emboli were probably related to the use of a thick walled catheter with side holes. In order to manipulate the catheter into the left common carotid artery a spring guide wire had been inserted into the catheter. During the several minutes required for this maneuver the catheter could not be adequately flushed. Inspection of the catheter following its withdrawal suggested that clots had formed in the side holes. Because of slight narrowing of the diameter of the side holes toward the lumen of the catheter subsequent aspiration was unlikely to dislodge them but they could be ejected by the force of a vigorous hand injection.

Discussion

During cerebral angiography by either direct percutaneous puncture or indirect catheterization of the carotid arteries embolic occlusions of small intracerebral arteries have been identified. In some cases a neurologic complication appropriate to the site of the occlusion resulted from the procedure. In other cases the patients were asymptomatic. Analysis of the cases suggested that the emboli were related to the angiographic technique. This presumption was supported by a decrease in the incidence of such complications when the technique was changed.

Embolic complications may have been unrecognized previously due to the difficulty in identifying small intracerebral occlusions. The validity of the criteria for occlusion have been confirmed by biopsy or autopsy finding of cerebral infarction in three cases not described in this report. Experimentally produced emboli in humans present a similar angiographic appearance (12). The demonstration of such occlusions may only be possible radiologically. Small arterial occlusions are difficult to identify at post mortem examination even when accompanied by infarction. Spontaneous lysis which may occur within hours to days of an occlusion may partially explain this difficulty (20).

In most of the cases in which embolic occlusions were identified, the films obtained during the actual study enabled the diagnosis to be established. In some cases in which a neurologic complication developing during angiography could not be explained by interference with blood flow in the carotid artery a repeat study was done immediately to determine the cause of the



Fig. 3 Case 3 a) and b) Mid arterial and early venous phases from a lateral series following percutaneous puncture of left carotid artery. In (a) the right anterior cerebral artery is filled and obscures the left pericallosal artery. In (b) the prolonged filling of the left pericallosal artery after the right anterior cerebral vessel had emptied indicates stasis and reveals the occlusion to be responsible. A filling defect is present at the origin of the callosal marginal branch and is indicated by the left open arrow. An abrupt termination of the pericallosal branch indicated by the right open arrow is another point of occlusion. This appearance persisted during multiple films. In (a) no branches of the middle cerebral artery are seen in the mid and posterior frontal regions. The solid arrow points to beginning filling in retrograde fashion of one of the peripheral branches of the occluded middle cerebral vessel. This branch is also indicated by a solid arrow in (b) where it is filled further toward the Sylvian fissure.

ter was introduced into the left carotid artery to a point approximately 2 cm below the bifurcation. A single injection of 8 ml of Renografin 60% was made by hand for a lateral film of the neck. Immediately after the injection it was noted that the patient was unresponsive to verbal stimuli and was aphasic. The catheter was immediately pulled back from the left carotid artery into the aortic arch. Her stretch reflexes were equal and no Babinski signs were elicited. There was withdrawal to pain in all four extremities. The blood pressure was unchanged. The patient was given 5% CO₂ in oxygen by face mask and her condition appeared to be stabilized. An attempt was made to reinsert the catheter into the left carotid artery but it was unsuccessful. During the manipulation of the catheter within the aortic arch it was noted that the patient had a right hemiparesis with a right Babinski sign and was less responsive to auditory stimuli than before. Review of the films of the neck vessels and the right intracerebral vessels revealed no significant abnormalities.

It was decided to continue the investigation by a different route in order to determine the cause of the stroke. A percutaneous left carotid puncture was made and lateral serial films of the skull were exposed. The films revealed occlusions in peripheral branches of both the anterior and middle cerebral arteries (Fig. 3). This procedure did not change the patient's neurologic status but her blood pressure decreased from 150/90 to 180/60. Within 24 hours there was considerable improvement in the right hemiparesis but the aphasia which was both receptive and motor persisted. Over the next 12 days the patient's mental status im-

proved slightly and she began to respond to verbal commands. At the time of discharge 13 days after the angiography the motor aphasia was present without evidence of the right hemiparesis.

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Fig. 3 Case 3 a) and b) Mid arterial and early venous phases from a lateral series following percutaneous puncture of left carotid artery. In (a) the right anterior cerebral artery is filled and obscures the left pericallosal artery. In (b) the prolonged filling of the left pericallosal artery after the right anterior cerebral vessel had emptied indicates stasis and reveals the occlusion to be responsible. A filling defect is present at the origin of the callosal marginal branch and is indicated by the left open arrow. An abrupt termination of the pericallosal branch indicated by the right open arrow is another point of occlusion. This appearance persisted during multiple films. In (a) no branches of the middle cerebral artery are seen in the mid and posterior frontal regions. The solid arrow points to beginning filling in retrograde fashion of one of the peripheral branches of the occluded middle cerebral vessel. This branch is also indicated by a solid arrow in (b) where it is filled further toward the Sylvian fissure.

ter was introduced into the left carotid artery to a point approximately 2 cm below the bifurcation. A single injection of 8 ml of Renografin 60% was made by hand for a lateral film of the neck. Immediately after the injection it was noted that the patient was unresponsive to verbal stimuli and was aphasic. The catheter was immediately pulled back from the left carotid artery into the aortic arch. Her stretch reflexes were equal and no Babinski signs were elicited. There was withdrawal to pain in all four extremities. The blood pressure was unchanged. The patient was given 5% CO₂ in oxygen by face mask and her condition appeared to be stabilized. An attempt was made to insert the catheter into the left carotid artery but it was unsuccessful. During the manipulation of the catheter within the aortic arch it was noted that the patient had a right hemiparesis with a right Babinski sign and was less responsive to auditory stimuli than before. Review of the films of the neck vessels and the right intracerebral vessels revealed no significant abnormalities.

It was decided to continue the investigation by a different route in order to determine the cause of the stroke. A percutaneous left carotid puncture was made and lateral serial films of the skull were exposed. The films revealed occlusions in peripheral branches of both the anterior and middle cerebral arteries (Fig. 3). This procedure did not change the patient's neurologic status but her blood pressure decreased from 150/90 to 180/60. Within 24 hours there was considerable improvement in the right hemiparesis but the aphasia which was both receptive and motor persisted. Over the next 12 days the patient's mental status im-

The decrease in the incidence of embolic complications during direct needle angiography when the replacement of the stylet was eliminated led us to believe that the emboli were most likely fragments of blood clot, it was not until after the incidence of emboli had decreased that a change was made to a closed system for flushing the needle. It is also likely that blood clots were the emboli in Case 3 since the occlusions produced by cotton fibers in the documented cases have been in vessels much smaller than those involved in this case.

It is obvious that the elucidation of the cause of angiographic complications is essential to the elimination of such complications. The detection of asymptomatic emboli may lead to changes in technic which will prevent more serious complications in other cases. For these reasons as well as others it is important to study the angiographic films for small vessel occlusions. Careful comparison between the films of different series and between the current study and any previous study may make small occlusions obvious.

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We are grateful to Dr Herbert L. Abrams, Professor of Radiology and Director of the Diagnostic Division, for his critical review of this manuscript.

SUMMARY

Three cases are reported in which significant complications of cerebral angiography were due to emboli. The emboli were thought to arise in the hub of the needle used in two cases and in the side holes of a catheter in the third. The technical means to avoid such complications are discussed. The detection of small intracerebral occlusions indicates the need to examine the technic used in order to eliminate the possibility of iatrogenic emboli. The cases reported also document the immediate availability of leptomeningeal anastomoses as a source of collateral supply to an occluded vessel.

ZUSAMMENFASSUNG

Drei Fälle mit signifikanten Komplikationen bei cerebraler Angiographie waren durch Embolie verursacht. Der Ursprung der Embolie wurde in zwei Fällen in der Injektionsnadel und in einem Fall in der seitlichen Öffnung eines Katheters vermutet. Es werden die technischen Mittel zur Verhinderung solcher Komplikationen besprochen. Die Entdeckung von kleinen intracerebralen Verschlüssen weist auf die Notwendigkeit hin, die angewendete Technik zu überprüfen, um das Risiko von iatrogenen Embolien zu beseitigen. Die berichteten Fälle beweisen auch das Vorhandensein von leptomeningealen Anastomosen, wovon die Kollateralkirkulation zu einem verschlossenen Gefäß ausgeht.

RÉSUMÉ

Présentation de trois cas où des complications graves de l'angiographie cérébrale étaient dues à des embolies. Les auteurs pensent que les embolies se sont formées dans la

complication. This did not appear to increase the severity of the neurologic complication.

While blood clots originating from the needle are mentioned as a possible source of emboli in discussions of complications, this has not often been documented (5, 16). Emboli to the retina as a complication of angiography have in general been thought to be associated with atherosclerosis (1, 8, 13, 15). LEVINE & HENRY (11), however, have reported retinal embolization following carotid angiography without evidence of underlying arteriosclerotic disease.

It is not possible to establish how often embolic complications occur during catheter angiography. LANG reported 9 cases of arterial embolization in a review of 11,402 percutaneous angiograms (9). LÖDLING & ÖVENFORS have found small infarcts in dogs after selective renal arterial catheterization which they attributed to blood clots (6, 7). It is likely that most small emboli are asymptomatic in viscera other than the brain and are consequently undetected.

BOIJSEN & FEINSTEIN discussed the potential hazards of catheters with side holes, such as the formation of emboli from clots (2). They suggested that this type of catheter should be avoided for elective examinations except in angiocardiology. OLIN has reported that clots are more likely to form in side holes in thick walled radio opaque catheters than in thin walled non opaque polyethylene catheters and he has emphasized the necessity for vigorous flushing of the catheters while they are in place (14). At the present time we limit the use of such catheters to placements within large vessels that do not require prolonged manipulation and we exclude their use from any selective studies of the cerebral vessels. With the increased use of catheter techniques for the study of cerebral vessels, particularly in the older age groups, careful attention to technical problems will be necessary to avoid embolic complications.

It is possible that the emboli may have been foreign material such as cotton fiber particles. Such particles may arise either from wiping the needle, stylet, or wire leader with gauze containing short cotton fibers or from using solutions contaminated with cotton particles to flush the needle or catheter (1, 3, 17). Our pathologist has found evidence of such emboli in specimens obtained after selective renal catheterization and direct needle carotid angiography. Experimental work in animals in our laboratories has confirmed the large number of potential embolic particles that can accumulate in open bowls (1). It is now routine in all of our vascular studies to use a closed system for flushing solutions. This is conveniently done by using a standard commercial intravenous solution and tubing set which is connected to a stopcock on the catheterization tray. The solution is drawn into the syringe used for flushing by connecting it to the stopcock.

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It is obvious that the elucidation of the cause of angiographic complications is essential to the elimination of such complications. The detection of asymptomatic emboli may lead to changes in technic which will prevent more serious complications in other cases. For these reasons as well as others it is important to study the angiographic films for small vessel occlusions. Careful comparison between the films of different series and between the current study and any previous study may make small occlusions obvious.

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Three cases are reported in which significant complications of cerebral angiography were due to emboli. The emboli were thought to arise in the hub of the needle used in two cases and in the side holes of a catheter in the third. The technical means to avoid such complications are discussed. The detection of small intracerebral occlusions indicates the need to examine the technic used in order to eliminate the possibility of iatrogenic emboli. The cases reported also document the immediate availability of leptomeningeal anastomoses as a source of collateral supply to an occluded vessel.

ZUSAMMENFASSUNG

Drei Fälle mit signifikanten Komplikationen bei zerebraler Angiographie waren durch Embolie verursacht. Der Ursprung der Embolie wurde in zwei Fällen in der Injektionsnadel und in einem Fall in der seitlichen Öffnung eines Katheters vermutet. Es werden die technischen Mittel zur Verhinderung solcher Komplikationen besprochen. Die Entdeckung von kleinen intracerebralen Verschlüssen weist auf die Notwendigkeit hin, die angewendete Technik zu überprüfen, um das Risiko von iatrogenen Embolien zu beseitigen. Die berichteten Fälle bezeugen auch das Vorhandensein von leptomeningealen Anastomosen, wovon die Kollateralkirkulation zu einem verschlossenen Gefäß ausgeht.

RÉSUMÉ

Il est présenté on de trois cas où des complications graves de l'angiographie cérébrale étaient dues à des embolies. Les auteurs pensent que les embolies étaient dues à des fragments de caillot sanguin. Dans deux cas, les embolies étaient dues à des fragments de caillot sanguin qui s'étaient formés dans le hub de la aiguille. Dans le troisième cas, les embolies étaient dues à des fragments de caillot sanguin qui s'étaient formés dans les trous latéraux d'un cathéter. On discute les moyens techniques pour éviter de telles complications. La détection de petites occlusions intracérébrales indique le besoin d'examiner la technique utilisée afin d'éliminer la possibilité d'embolies iatrogènes. Les cas rapportés documentent également la disponibilité immédiate des anastomoses leptoméningées comme source d'approvisionnement collatéral pour un vaisseau occlus.

l'aiguille utilisée dans deux cas et dans les trous latéraux du cathéter dans le troisième cas. Ils examinent les moyens d'éviter ces complications. La découverte de petites obstructions artérielles intracérébrales rend nécessaire l'examen de la technique utilisée pour éliminer la possibilité d'embolies iatrogéniques. Les cas présentés montrent aussi le fonctionnement immédiat d'anastomoses leptoméningées pour l'irrigation collatérale d'un vaisseau obstrué.

REFERENCES

1. ADAMS D., OLIN I. and ROSEK J. Cotton fiber embolization during angiography. *Radiology* 81 (1965), 678.
2. BOIJSEN E. and FEINSTEIN G. L. Arteriographic catheterization technic. *Am J Roentgenol* 85 (1961), 1037.
3. CHASON J. L., IANDERS J. W. and SWANSON R. E. Cotton fiber embolism. A frequent complication of cerebral angiography. *Neurology* 13 (1963), 558.
4. DAVID N. J., KLINTWORTH G. K., IRLBERG S. J. and DILLON M. Fatal atheromatous cerebral embolism associated with the appearance of bright plaques in the retinal arterioles. *Neurology* 13 (1963), 708.
5. EININ M. and GORNSEN J. Complications of carotid angiography in acute cerebrovascular conditions. *Acta Med scand* 172 (1962), 151.
6. EDLING N. P. G. and OVENFORS C. O. Risks in selective renal catheterization and arteriography. *Acta radiol* 2 (1964), 241.
7. — — Intentional embolism in selective renal arteriography. *Acta radiol* 2 (1964), 316.
8. HANEY W. P. and PRESTON R. F. Ocular complications of carotid arteriography in carotid occlusive disease. *Arch Ophthalmol* 67 (1962), 127.
9. LANG E. K. A survey of the complications of percutaneous retrograde arteriography. *Radiology* 81 (1963), 257.
10. LANNER L. O. and ROSENGREN K. Angiographic diagnosis of intracerebral vascular occlusions. *Acta radiol* 2 (1964), 129.
11. LEVINE R. A. and HENRY M. D. Ischemic infarction of the retina following carotid angiography. *Am J Ophthalmol* 55 (1963), 363.
12. LUESSENHOP A. J., GIBBS M. and VELASQUEZ A. C. Cerebrovascular response to emboli. *Arch Neurol* 7 (1962), 264.
13. MCBRIEN D. J., BRADLEY R. D. and ASHTON N. Nature of retinal emboli in stenosis of the internal carotid artery. *Lancet* 1 (1963), 697.
14. OLIN I. Studies in angiographic technique. p. 96. Håkan Ohlssons Boktryckeri, Lund, 1963.
15. ROSS R. R. W. Atheromatous retinal embolism. *Lancet* 2 (1963), 1354.
16. SCHEINBERG P. and ZUNER E. Complications in direct percutaneous carotid arteriography. *Arch Neurol* 8 (1963), 676.
17. SILBERMAN J., CRAVIOTO H. and FEIGIN I. Foreign body emboli following cerebral angiography. *Arch Neurol* 3 (1960), 711.
18. TAVERAS J. M. Angiographic observations in occlusive cerebrovascular disease. *Neurology* 11 (1961), 86.
19. WICKBOM I. Survey of the angiographic technique used in patients suspected of cerebral ischemia. *Ann Radiol* 6 (1963), 23.
20. ZATZ L. M., IANNONE A. M., ECKMAN I. B. and HECKER S. P. Observations concerning intracerebral vascular occlusions. *Neurology* 15 (1965), 389.

